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WOUND SURGERY ENCOUNTERS A DILEMMA

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The object of this Society shall be to encourage investigations of the physiology and pathology of the thorax and its contents, to stimulate the study of the etiology, symptomatology, pathology and treatment of diseases affecting the thorax and its contents, and to disseminate such knowledge. (Article II, Section 1. Constitution of The New York Society for Thoracic Surgery.)

IF MICHELANGELO had fallen from the scaffolding while decorating the ceiling of the Sistine Chapel it is likely that he would have been treated by John of Vigo. John of Vigo was the first surgeon to record his views on gunshot wounds of the chest, and in fact his book *Practica Copiosa* printed in Rome in 1514 was one of the very early surgical texts to mention wounds by firearms. John of Vigo enjoyed a rich experience in wound surgery while attached to that fighting Pope, Julius the Second, and a nice legend perpetuated by Sir Clifford Allbutt places a French translation of the *Practica* in the hands of the young barber's apprentice, Ambroise Paré. At any rate, Paré, in 1575, quoted John of Vigo on the management of wounds of the chest.

At first thought it might seem that the treatment of an open pneumothorax would have been a straightforward matter to the ancient wound surgeons. Such a wound presents what might appear to be a simple choice between two procedures: the wound can be closed or it can be left open. As a matter of fact, the several factors which are determinants to this decision puzzled surgeons for centuries and still crop up in one form or another in our own century. In my title I have referred to this choice as a dilemma and propose to identify some of the circumstances which made the choice between leaving the wound open and closing it a difficult one.

Address at the Fortieth Anniversary Dinner of The New York Society for Thoracic Surgery, November 8, 1957.

Paré identified two major considerations. "John of Vigo," he wrote, "said that there is a disagreement between the surgeons because some have a mind to close the penetrating wound as soon as possible . . . for fear that the cold air would penetrate into the heart and that vital spirits would depart and vanish; the others, contrary minded, recommend keeping the wound open . . . in order to evacuate the blood left in the thorax, fearing that this would decompose and putrefy; that would provoke fever, fistula and other complications. In truth, those who are of the mind to close the wound immediately . . . are entirely right, on condition that there is no blood or only a small amount inside the chest. . . . In the same manner are those quite right who have the mind to keep the wound open . . ."

It is significant that Paré set out to define certain conditions or principles which were to distinguish between the two courses of action and point to the selection of the one better adapted to the particular circumstances. His text was written for the benefit of barber surgeons who, as workers in a practical art, desired to be guided by definite rules and precepts, preferably phrased in the imperative. They expected to be told either "leave the wound open" or "close the wound." Such workers are particularly unhappy when the exercise of choice between alternative methods of procedure rests on conditions that they themselves may be unable to recognize. The condition which determined that the wound should be left open was indeed definite even though difficult to assess—blood in the chest. The concept of what would happen if blood were closed in the chest was straightforward and empirical. The blood would "decompose and putrefy" and would "provoke fever, fistula and other complications."

Paré was on less certain ground when he attempted to define the conditions under which the wound should be closed immediately. He may have known that he was on unfamiliar ground, for he repeated the caution that there must be no blood or at least only a small amount of blood inside the chest if this course were to be elected. Then departing from his own surgical experience, Paré invoked a prevailing doctrine of ancient authority. "Close the wound," he wrote, "for fear that the cold air will penetrate into the heart and the vital spirits depart and vanish."

So the dilemma presented by an open chest wound as seen by the great surgeon of the Renaissance was: surgeons who close the wound to keep the vital spirits from departing may be right but experience shows that if blood remains in the chest, mischief will follow. Judging from the case reports of his own practice, it was Paré's custom to keep the wound open 2 or 3 days and allow the blood to trickle out by gravity; when the flow of blood ceased, the wound was closed. This compromise undoubtedly reflects Paré's estimate of the relative validity of the indications for the choice between closing the wound and leaving it open under the circumstances which actually prevailed. It is likely that the wounded of that period who survived to come under treatment by a surgeon had wounds of small cross-sectional area resulting from sword thrusts or low velocity missiles.

Let us now briefly trace the development of the more precise understanding of the two courses of action that confronted Paré as the two horns of a dilemma, first taking up the matter of blood in the pleural cavity. The untoward effects of the accumulation of blood in any wound are related in the legends of ancient wars, and many romantic stories can be found of friends who, having put their months to the flesh, sucked blood from the wounds of warriors. In the wars of the seventeenth and eighteenth centuries the sucking of wounds was so much in demand that it became a trade. Certain individuals in a regiment, frequently the drummer, became famous for their skill as wound suckers. There is adequate testimony in the old writings to establish the efficacy of this form of treatment, which of course is in principle a sound method in the light of present-day knowledge. Sucking was found particularly useful when quarrels were decided by duels with swords which terminated with the first flesh wound. The wounded swordsman after this form of treatment commonly was able to walk home and entirely conceal the affair. Today it is recognized that contaminated blood remaining in a wound track offers an inviting pabulum for the growth of pathogenic bacteria, and it is the usual practice with flesh wounds to leave them open with textile drainage and to close them on the fourth day by reparative suture.

The activities of wound suckers finally fell into disrepute and the procedure itself became known as the *secret dressing*. To inflate their prestige and possibly in order to keep their trade to themselves the suckers pretended to make it a magical ceremony. They muttered cabalistic words, made strange motions, and drew the sign of the cross. It was understandable that the priests who also followed armies became outraged by such profanities and refused extreme unction or other sacrament to those who participated in these diabolical ceremonies. The suckers on the other hand refused to ply their art on those who had commerce with the priests under the pretense that Christian rites interfered with their incantations. La Motte, a surgeon of this period, was an eye witness to many wonderful cures. He saw for example, a soldier who had been run through the breast with a single lunge which passed, as he described it, "in at the pap, and out at the shoulder." By examining the wounds and noticing the length of the antagonist's sword La Motte was well satisfied that the weapon had piereed the lungs and gone quite across the chest. The drummer of the regiment first sucked one wound, then turned his patient and sucked the opposite wound. He applied a piece of chewed paper upon each and next day the soldier was seen walking in the streets.

Wound sucking was thought to be particularly effective for wounds of the chest although some writers expressed doubt that retained blood actually could be removed from the pleural cavity. The question can be asked whether the phrase "sucking wound" as it is applied today to open chest wounds is a curious linguistic survival from this period because actually such a wound blows air out as much as it sucks air in.

In 1707, Anel[1] published in Amsterdam a small book on the art of sucking wounds without using the human mouth. A silver tube was the answer. One end could be attached to a piston syringe and the other applied to a wound. By

this gadgetry the surgeon himself was able to take over the procedure of wound sucking, which then became known by the more refined term—aspiration. Improvements in the apparatus inevitably followed and one of these proposed by Birkholz,[2] in 1771, introduced a reservoir into the suction line, producing in essence a Potain aspirator. Anel doubted that suction applied to the margins of the wound could actually evacuate the pleural cavity and so designed a cannula patterned after a female catheter which could be introduced into the pleural space through the wound. From this it was a short step to trocar and catheter.

In 1772, Valentin[3] published his small volume in which he declared that all wounds of the chest were to be closed as a measure to suppress hemorrhage and that when necessary a counter opening should be made to evacuate the effused blood at an early date. Guthrie,[4] the great English military surgeon of the Napoleonic Wars and the Peninsular Campaign, closed wounds of the chest but followed them carefully with what he called "unremitting attention in the use of the stethoscope," and removed blood by trocar and cannula. "The first object," Guthrie wrote, "is to save life. After that, if time be given, the next will be to relieve the loaded cavity." Baron Larrey,[5] Napoleon's surgeon, supposed that 9 days should elapse before it was safe to evacuate blood without encountering the danger of renewed bleeding. If Guthrie, surgeon of the Duke of Wellington, could find an excuse to differ from Larrey he always did so; his recommended procedure was to wait until the patient reacted, when he concluded that bleeding had ceased. In following the situation he looked for the increase of serous effusion which usually came from the third to the fifth day. Then Guthrie either reopened the wound to evacuate blood or carried this out through another opening with a trocar and cannula. He placed great importance on relieving the lung from pressure at an early date so that it might expand and not be bound down by what he termed false membranes.

Having traced these significant steps in the management of the retained blood, let us examine briefly the other horn of the dilemma, namely the disturbance in pulmonary ventilation which attends an open pneumothorax. In the view of Paré, disaster resulted from the cold air penetrating into the heart and the vital spirits departing the body through the wound and vanishing. Here, as with the complication of blood accumulating in the wound, the record of experience is lost in antiquity. Take the famous case of The Dying Gaul in the Capitoline Museum of Rome dating from the second or first century B.C. (Fig. 1). Blood is pouring from the wounded chest of the warrior and his head sinks forward as his eyes dim with hypoxia. The breathing is labored and he supports his weight on a rigid arm to obtain maximal use of the unwounded lung.*

Little was understood about the lethal effects of an open pneumothorax until the early physiologists started a systematic analysis of the action of the organs of respiration. The phenomenon of pendulum air in open pneumothorax, a phenomenon which is customarily attributed in eponym to Brauer, was explained by Hoadley[6] as early as 1737.

*Elsewhere I have given due credit to Dr. Evarts Graham for this clinical diagnosis.

> "In expiration, the air in the unwounded side was condensed, and part of it, instead of going out at the windpipe, forced its way into the lung of the wounded side, and dilated it, till the air within it came into an equilibrium with the external air which surrounded it; and in inspiration, when the air in the lung of the unwounded side became rarer than the external air, the lung in the wounded side was compressed, and part of the air within it was, by the pressure of the external air, forced back into the lung of the unwounded side, till the equilibrium was again restored."

On the basis of clinical observations, the brilliant young associate of the Hunters, William Hewson,[7] observed in 1767 that with an open external wound the patient may hardly be able to breathe but he breathes easily when it is covered. Hewson said the difficulty arose "owing to the air getting into the cavity of the thorax in inspiration instead of entering the lungs by the trachea."

Fig. 1.

While such a wound is uncovered the patient is deprived of the use of the lobes of that side either partially or entirely "according as the wound of the thorax bears a less or greater proportion to the branch of the trachea to that side."

These concepts based both on physiologic experiments and on clinical observations in the eighteenth century stand today except for certain amplifications and minor corrections.

It is difficult to be certain when wound surgeons closed a defect in the chest wall primarily to reestablish effective pulmonary ventilation rather than to control hemorrhage from the lung. The following story related in the memoirs

of Baron Larrey, the surgeon of Napoleon, emphasizes the immediate and striking improvement in the condition of the patient that follows the closure of an open chest wound.

> "A soldier was brought to the hospital of the fortress of Ibrahym Bay, immediately after a wound . . . that penetrated the thorax, between the fifth and sixth true ribs, . . . it was about eight centimetres in extent: a large quantity of frothy and vermilion blood escaped from it with a hissing noise, at each inspiration. His extremities were cold, pulse scarcely perceptible, countenance discoloured, and respiration short and laborious; in short, he was every moment threatened with a fatal suffocation.
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> "After having examined the wound, and the divided edges of the parts, I immediately approximated the two lips of the wound, and retained them by means of adhesive plasters, and a suitable bandage round the body.
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> "In adopting this plan, *I intended only to hide from the sight of the patient and his comrades, the distressing spectacle of a hoemorrhage,** which would soon prove fatal: and I therefore thought, that the effusion of blood into the cavity of the thorax, could not increase the danger.
>
> "But the wound was scarcely closed, when he breathed more freely, and felt easier. The heat of the body soon returned, and the pulse rose; in a few hours he became quite calm, and to my great surprize, grew better. He was cured in a very few days, and without difficulty."

Larrey then referred to two similar cases at the hospital of the Imperial Guard.

> "Since Ambroise Paré, all practitioners and authors, who have written on wounds of the thorax, advise not to close the wounds which penetrate this cavity, especially, when they are followed by hoemorrhage. . . . But the ancients closed such wounds, and even used sutures to make them more exact. . . .
>
> "The surprising success that attended these three apparently mortal wounds . . . led me to believe that this practice is preferable to that which is now adopted."

Guthrie, whom I have already identified as the surgeon of the Duke of Wellington, remarked that Baron Larrey had received credit for advocating closure of chest wounds merely because he had so effectively supported the views of Valentin, and that although the precepts of Valentin may have been forgotten in France they were in existence and Larrey merely deserved the merit of having revived and confirmed them. Furthermore, Guthrie claimed that he himself had closed chest wounds long before he had seen any of Larrey's works and was not aware that he was acting out of the ordinary course of things except that he used a continuous suture instead of trusting to a compress and bandage. While Guthrie pointed to this as a trivial difference, far more weight would be put on this detail today, because until a surgeon can be certain that a wounded lung is not leaking air into the pleural space, the

*Italics are those of the author.

external wound should not be closed tightly with a suture. A lethal pressure pneumothorax may develop within a few minutes. Strapping the wound edges together as Larrey did permits the escape of air should pressure build up from a bronchopleural fistula.

It was well known to Guthrie that closure of chest wounds relieved difficult breathing and he was familiar with the observations of Hewson to which he referred. Nevertheless his writings give the impression that his advocacy of closure of the wound was based primarily on the belief that in accord with the recommendation of Valentin this was the most efficacious means of arresting hemorrhage from the lung. Thus the two horns of Paré's dilemma became a point of issue between the surgeons of Napoleon and Wellington at Waterloo.

There has been constant modification and duplication of detail in devices designed to suppress the in-and-out flow of air in an open chest wound. Hermetical seals were tried in the Northern armies of the Civil War.[8] Tampons were designed in World War I[9] and, on the Italian front, Morelli[10] used his occlusive rubber cuffs on tubes permitting suction drainage as well as closure of the defect.

Further projection of the development of the understanding of open pneumothorax would lead directly into the thinking and experiments which eliminated the hazard of uncontrolled open pneumothorax during a chest operation. This is a long and fascinating story of surgical technology and invention in which members of this Society and their preceptors have played an active part. I mention specifically Willy Meyer, Nathan Green, O'Dwyer, Meltzer, Auer, and Lilienthal. The devices developed to maintain ventilation of the lungs in the patient undergoing thoracotomy were many and varied. Today the responsibility for ventilation has been assumed by the anesthetist, but for several decades this responsibility was added to that of the operation itself and assumed by the surgeon. Rather than pursue the intricacies of this story,[11] it may be of greater interest to review some of the circumstances that attended World War I and surrounded the task of the Empyema Commission of 1918.

A beginning may be made in 1914, 3 years before the United States entered the War. At that time a fairly complete concept of the aerodynamics of pulmonary ventilation had been established; methods to maintain adequate ventilation during open chest operations had been devised, and techniques for applying these methods had been perfected. It was well into 1916 before the treatment of gunshot wounds of the chest by open operation in the combat zone had been undertaken except in the German army. Both French and British surgeons were submerged by an overwhelming load of casualties and preoccupied with the task of learning by bitter experience the surgical principles that must guide the management of flesh wounds from high velocity missiles and high explosive shell fragments.

Then the Parisian surgeon, Pierre Duval,[12] spearheaded what appeared to be a break-through on the surgical front.

> "Surgery of the lung has undergone complete revolution in the war. The lung is no longer an organ inaccessible to the surgeon. One can open the chest widely, expose the lung, lobe by lobe, manipulate it, resect it, suture it, and treat it as one would a coil of intestine in a laparotomy. Surgery of the lung does not require any of those pressure chambers which the genius of the Germans invented and their persuasian made us think necessary."

It is only fair to point out that Pierre Duval's claims were not widely accepted in France, where Grégoire and Courcoux maintained a far more conservative position. British surgeons were also skeptical although Moynihan and Gask were swept along on the tide of enthusiasm. H. Morriston Davies with his scholarly interest in thoracic physiology held to sound concepts, as did William Hutchinson of the Canadian army.

The rash claims of Duval and his disciples and their scorn of differential pressure had more repercussions in the United States than in the theater of war. In the general exuberance over all things French and the condemnation of all things German, the fact was forgotten that scientific concepts recognize no national boundaries. Lord Moynihan,[13] then Sir Berkeley, Colonel in the R.A.M.C., informed audiences in the United States that a surgeon might attack the lungs boldly, without heed to the danger of an open pneumothorax. Following the pattern of the triumphal tours of the victorious generals, Duval appeared before professional groups in the United States in the autumn of 1918. I quote from an Editorial[14] in the American Journal of Surgery:

> "Those who were fortunate enough to hear the addresses of the visiting foreign surgeons at the College of Physicians and Surgeons and at the New York Academy of Medicine in the early November days will not soon forget these memorable occasions. They were all inspiring addresses, especially in the setting of spontaneous patriotic fervor that marked those days. They were set forth with a remarkable lucidity and grace of diction; charged with big and novel surgical principles and bearing a message of profound import not only to war surgery, but to surgery of civil life.
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> . . . we have selected for comment in this number their experiences in lung surgery, because this was perhaps the most startling feature in their addresses. This phase was beautifully set forth by Major Duval, the daring originator of the latest treatment of wounds of the chest and lung . . . no one could accuse Duval of chauvinism when he pointed out that certain German principles, such as the teaching of the exaggerated dangers of artificial [sic] pneumothorax . . . had retarded the progress of lung surgery appreciably . . . the lung, like other tissues of the body, need not be handled with the timidity with which it had hitherto been accustomed. . . . Essentially, therefore, the method devised by Duval consists in wide exposure of the chest cavity, producing a complete pneumothorax with prolapse of the lung; pulling out and exposing each lobe of the organ, and treating the wound in the same manner as a wound in any other part of the body, followed by complete closure of the thoracic cavity, without drainage."

And so patriotic fervor once again was to solve both horns of the old dilemma of John of Vigo and Paré.

In reflecting upon the narrative of the experience of wound surgeons in the 1914-1918 War as compared with that of surgeons in previous and subsequent wars, I have become increasingly convinced that the outstanding feature of this period was the prevalence of hemolytic streptococcus infection both in Europe and in the United States. This epidemic assumed pandemic proportions when the hemolytic streptococcus rode the coattails of the influenza virus which swept around the world. For over a decade after the end of the war the hemolytic streptococcus haunted the surgical wards of our civilian hospitals and made return visits, particularly during the winter months. Those of us studying surgery during this period were weaned on the surgery of acute tenosynovitis, mastoiditis, primary streptococcal peritonitis, erysipelas, and the catastrophic infections of supposedly aseptic operative fields with this organism. The streptococcal pneumonia and empyema which accompanied the influenza epidemic of 1918 was a new disease to a generation of doctors. They had no precedents which defined it. In the past three decades, as far as my own experience is concerned, the disease is, for all practical purposes, extinct. During the 1914-1918 War, however, it gave rise to the Empyema Commission, which in the minds of thoracic surgeons is identified with Dr. Evarts Graham.

Following the entrance of the United States into World War I, Dr. Graham* was on duty as a captain in the Medical Corps assigned to take a course in neurosurgery in Chicago when he was visited by Dr. Allen Kanavel who was on duty as a consultant in the Office of the Surgeon General of the Army. Kanavel told Graham that there was growing apprehension about difficulties ahead with empyema as the country was then in the first year of an influenza epidemic which would undoubtedly increase in severity—would he be interested in working on the problem? Graham knew little or nothing about chest surgery at that time but was interested in nitrogen loss from massive exudation and had been selected because of this interest and his chemical training. Shortly after Kanavel's visit, Graham received orders to Camp Lee, there to team up with Dunham, who was working on the bacteriology of the disease. Graham and Dunham were joined by Captain Bell, a chemist of the Sanitary Corps who died a few years after the war. These three formed the hard nucleus of what became known as the Empyema Commission. With the help of Kanavel, a questionnaire was issued to camp hospitals and it was found that the average mortality rates attributed to streptococcal empyema were running approximately 30 per cent. The surgical staffs of the camp hospitals were stubbornly antagonistic to members of the Commission, whom they regarded as "snoopers" from the Office of the Surgeon General. Opportunities for observation, however, could not be denied and disclosed that cyanotic patients, with pulmonary reserves crippled by massive and oftentimes bilateral bronchopneumonia, were being hurried to an operating room as soon as thoracentesis yielded fluid containing chains of streptococci. The operation was rib resection with open tube drainage. Death occurred quite regularly about a half an hour after the operation.

*This account of the formation and activities of the Empyema Commission was related to me by Dr. Graham.

It will be recalled that Sir Berkeley Moynihan had in the autumn of 1917 delivered the address in which he repeated the claims of Duval that the chest cavity could be opened as readily as the abdomen. When Graham, during his visits to army camp hospitals, expressed apprehension about the open pneumothorax which attended rib resection for empyema he was asked who he thought he was to contradict the great Moynihan. When Graham cited an actual case in which a cyanotic and dyspneic soldier had died on the operating table that very morning and asked if the open pneumothorax might not have been a contributing factor, the reply was: "No, he died because we didn't operate soon enough."

Early evacuation of streptococcal pus was taught as a dogma of the surgery of the period; tendon sheaths were opened on the least suspicion of infection and oftentimes a spreading streptococcal cellulitis was slashed with multiple incisions long before fixation abscesses appeared. Graham was certain that the open pneumothorax was a factor in the deaths from empyema but at that time was not familiar with the literature on open pneumothorax, nor did he have sufficient observation or experiments to prove his point.

In 1890, Bülau[15] had advocated drainage of empyema by the "closed" catheter technique, and the relative merits of closed drainage and open drainage by rib resection had been widely discussed in German-speaking countries. Emphasis, however, had been placed on the prevention of retraction of the lung and the quick healing which might be obtained, not on the immediate hazards of open pneumothorax nor on the reduction of functional lung volume to a point below tidal air requirements merely by the admission of air to the pleural space.

In December, 1918, Evarts Graham[16] wrote:

> "During the past year the idea has become prevalent in this country that operations upon the thorax which permit of the free entrance and exit of air into the pleural cavity during the operation can be performed with about the same impunity as abdominal operations. In fact, the belief has arisen that surgeons at the front have found that alterations of the intrathoracic pressure by the admission of air through extensive incisions or large gaping wounds are in themselves not dangerous, and a feeling of optimism exists that now bold surgical intervention on the thorax may be carried out. It has been said consequently that the danger from creating an open pneumothorax in early operations for empyema with permanent drainage is negligible."

Despite the developments and inventions and increased understanding of three and a half centuries, the army surgeons of 1918 were caught off balance when confronted by the old dilemma of Paré. The same problem presented itself in a slightly different form: whether to accept an open pneumothorax with the danger that the vital spirits would depart and vanish or to strive for a closed chest cavity with its hazards of infection and increasing encroachment on pulmonary ventilation by fluid and air in the pleural space. The problem was solved in the same ways that were devised by those who followed Paré—the insertion of a catheter and evacuation of the pleural cavity by "closed

drainage'' (Bülau); or repeated aspiration with needle and syringe until pleural adhesions localized an abscess that could be drained by rib resection without retraction of the lung. In the words of Guthrie, the first object became that of saving life; after that, if time were given, the next was to relieve the loaded cavity.

This display of ignorance of the principles that form the foundation of thoracic surgery annoyed and dismayed the few surgeons in this country who were familiar with the progress which had been made in Germany prior to the 1914-1918 War. In his Presidential Address of 1922,[17] Samuel Robinson spoke scornfully of those who would apply their war experience to that bugaboo of civilian surgery—bronchiectasis.

> ''It has always been my belief that the greatest triumph in thoracic surgery will be the surgical eradication of this deplorable disease. The enthusiast returns from the war who has often dragged a lung lobe into a spread thoracic wound, opened it, scraped it, washed it—yea, even removed it—and concluded, therefrom, that intrathoracic surgery is freed of its supposed dangers, and that the possibilities therein are comparable to those in the abdomen. Let him attempt the same performance in his home hospital in a case of lower lobe bronchiectasis. Then will he learn what real thoracic pathology means.''

There follows a vivid word picture of the surgical undertaking of lobectomy which ends abruptly with the ominous and cryptic statement: ''Suddenly, it is obviously time to return the patient to his bed. Not much has been accomplished.''

Samuel Robinson,[18] an advocate of *überdruck,* had introduced the positive pressure cabinet for thoracic operations at the Massachusetts General Hospital in 1909. Dr. Willy Meyer[19] worked with a chamber at the German Hospital in this city in the same year. This universal chamber combined the principles of *überdruck* and *unterdruck* and allowed for a wide range of pressure conditions. Both Robinson and Meyer were scholars of the surgical literature and both were in close touch with the progress in Germany.

It was against this background that your association which is tonight celebrating its fortieth anniversary was organized. By the article of its Constitution which appears as the motto of this address, this Society has brought light to the surgeons of twentieth century America as did John of Vigo and Paré to their colleagues of the Renaissance.

Much of the subject matter of this address was presented in the Mitchell Banks Lecture at the University of Liverpool on November 3, 1948, but not published.

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A NEW CLASSIFICATION FOR COARCTATIONS OF THE AORTA

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SINCE the first case report of a coarctation of the aorta was presented by Paris[1] in 1791, no adequate system or method of classification has been devised. Bonnet,[2] a French pathologist, in 1903 described the present commonly used system of classification of these lesions calling one an "adult" type and the other and "infantile" type. Most writers[3-7] on this subject have criticized this system, usually for more than one reason. In general the reasons have been: the incompleteness of the system, the omission of the rarer forms of the anomaly, no indication of or incomplete meaning denoted relative to the anatomy, pathology, or pathologic physiology of the particular situation.

We agree with the general feeling on the subject so well phrased by these writers. Bonnet's use of the terms "adult" and "infantile" is misleading. An adult may have the "infantile" variety or an infant may have the "adult" form. Burford[6] showed his appreciation of the problem when he suggested classifying these lesions on the basis of the location and patency of the ductus arteriosus. This was a well-justified improvement from the physiologic standpoint, but still leaves something to be desired. A similar situation prevails with the terms Bahnson[5] prefers and has advocated for these lesions. He called one type "uncomplicated coarctation" and the others "complicated coarctation." The classification offered by Edwards[7] attempted to encompass the various possibilities but it, too, has omitted certain prominent anatomic lesions while accentuating lesser associated anomalies or pathologic findings.

The lack of clear understanding and absence of unanimity of opinion as to the underlying factors concerned with the causation of the condition and the mechanism of development of aortic coarctation provide no etiological leads to a fully acceptable classification. Necessarily, recourse must be had to those anatomic and physiologic features which permit clinical differentiation of the several variants of this lesion.

In response to the clearly defined need for a completely new method of classification of these lesions, one of us (W. M. L.) has devised a system which we believe to be superior to any evolved thus far, and one which probably is acceptable clinically. This system was designed from the standpoint that the coarctation is the primary lesion, but gives consideration to the existence of intimately associated anomalies. Remotely associated congenital malformations and those occurring elsewhere are excluded by definition.

From the Hahnemann Medical College and Hospital, and the Bailey Thoracic Clinic, Philadelphia, Pa.
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TABLE I. CLASSIFICATION FOR COARCTATIONS OF THE AORTA

Type I	Coarctation of the aorta with	—ligament (ligamentum arteriosum)
Type II	Coarctation of the aorta with	—shunt, proximal or distal (patent ductus arteriosus
Type III	Coarctation of the aorta with	—hypoplasia
Type IV	Coarctation of the aorta with	—(isthmus) interruption
Type V	Coarctation of the aorta with	—site elsewhere (specify) a. ascending aorta b. aortic arch c. descending aorta (thoracic or abdominal) d. multiple coarctations

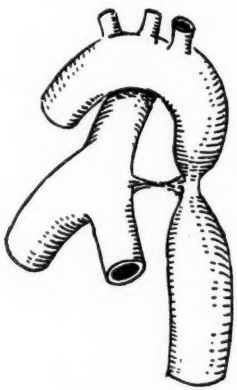


Fig. 1.—Type I. Coarctation of the aorta with ligament (ligamentum arteriosum).

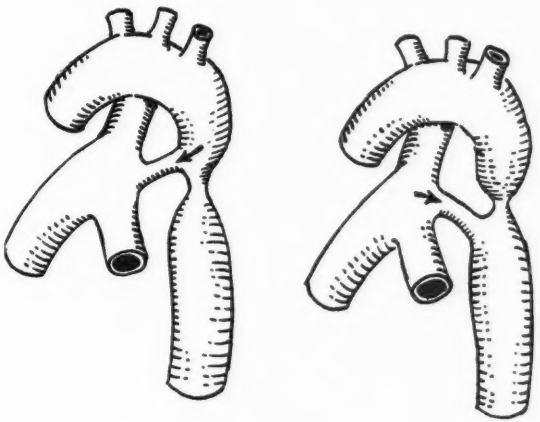
Type I. *Coarctation of the Aorta With Ligament.*—(Fig. 1.) According to Gross[4] this type encompasses fully 98 per cent of all coaretations of the aorta. This is the common type of lesion which is encountered just distal to the origin of the left subclavian artery in the course of the descending aorta. "With ligament" indicates that the ductus arteriosus has become obliterated and no longer is patent or capable of acting as a shunt. This being the case no importance can or should be attached to the point of attachment of this embryonic remnant to the great vessel. Its chief importance to the surgeon is as an aid in locating and dissecting out the left recurrent laryngeal nerve. These lesions usually involve but a very short segment of the aorta. However, those lesions which involve a longer segment but still occur in this region are included under this heading.

Type II. *Coarctation of the Aorta With Shunt, Proximal or Distal (Patent Ductus Arteriosus).*—(Fig. 2, *A* and *B*.) The location of the lesion under this heading is approximately the same as in Type I, but there are very important differences.

Whether the ductus is proximal or distal to the coarcted segment is significant. Those occurring proximal provide a passage for a left-to-right shunt of blood as a logical result of the application of the higher arterial pressure in

the aorta against the lower pulmonary arterial pressure. However, if the ductus arteriosus joins the aorta below the point of the aortic narrowing, the higher pulmonary artery pressure tends to produce a right-to-left shunt into the lower aortic segment. Thus, the lower portion of the body may be nourished largely by venous blood from the pulmonary artery. It is in these cases of right-to-left shunt that an observer may note cyanosis limited to that portion of the body which is below the crest of the ilium.

If the shunt is measured by catheterization and calculated in liters per minute, a number may be placed after the term "proximal" or "distal" to indicate the amount of the shunt.



A. *B.*

Fig. 2.—Type II. Coarctation of the aorta with shunt, proximal or distal (patent ductus arteriosus).

Theoretically there are two remaining situations which may occur in this type of anomaly which must be considered for the system to be complete. If for any reason the shunt should reverse itself from its usual (foregoing) course, the word "reversal" may be used followed by a number which indicates the amount of flow. Lastly, there is a possibility that the shunt, when measured, may be in "balance." To give an example:

Type II, (coarctation of the aorta with shunt), proximal, 1 L./min.

or

Type II, distal, 3 L./min.

or

Type II, proximal, reversed, 1.5 L./min.

or

Type II, distal, reversed, 2 L./min.

or

Type II, distal, balanced.

The various combinations of possibilities readily come to mind and the ease and simplicity with which they may be identified are apparent.

Type III. *Coarctation of the Aorta With Hypoplasia.*—(Fig. 3.) In hypoplasia, the aorta in most instances is of normal size up to the site of the pathologic lesion. Distal to it, the entire length of the great vessel is grossly diminished in size usually including its bifurcation and the iliac arteries. Once the involved coarcted segment, which usually is found at the site of Type I

Fig. 3.—Type III. Coarctation of the aorta with hypoplasia.

lesions and not long, has been excised and continuity re-established by an adequate anastomosis, the hypoplastic portion may prove to be an adequate passageway for blood flow. Associated anomalies of the left subclavian artery or an anomalous origin of the innominate or left common carotid artery may give rise to very unusual and interesting physical findings and radiologic observations, but have little to do with the classification of the basic lesions. Rarely, the hypoplasia may involve the aorta from its origin and include the coarctation.

We do not accept the idea that the lower pressure in the distal segment in itself is responsible for the small caliber of these uncommon cases. If the lower pressure and a decreased blood flow were responsible in them, hypoplasia also should be a most frequent finding in conjunction with Type I lesions.

Type IV. *Coarctation of the Aorta With Isthmus Interruption.*—(Fig. 4.) The isthmus of the aorta is that portion which lies just distal to the origin of the left subclavian artery and before the beginning of the descending thoracic portion of the artery. In the relatively long gap between the proximal and distal segments of the interrupted aorta there is, in most instances, a rudimentary remnant represented by a small fibrous strand of tissue which joins the two portions. Less commonly, this band may be lacking.

Invariably, in this condition, there is a large patent ductus arteriosus arising from the usual site on the pulmonary artery and inserting into the beginning of the distal segment of the aorta. Through this ductus, venous blood enters the lower segment to nourish the lower part of the body. Hence, cyanosis exists below the level of the iliac crests. These cases must be separated clinically from those of Type II with a distal shunt.

Fig. 4.—Type IV. Coarctation of the aorta with isthmus interruption.

Pathologically, Type IV coarctations may occur without the association of a patent ductus arteriosus, but this is an extremely uncommon form of the anomaly. We have seen only one instance of this situation and it was accompanied by multiple congenital anomalies which caused death soon after birth.

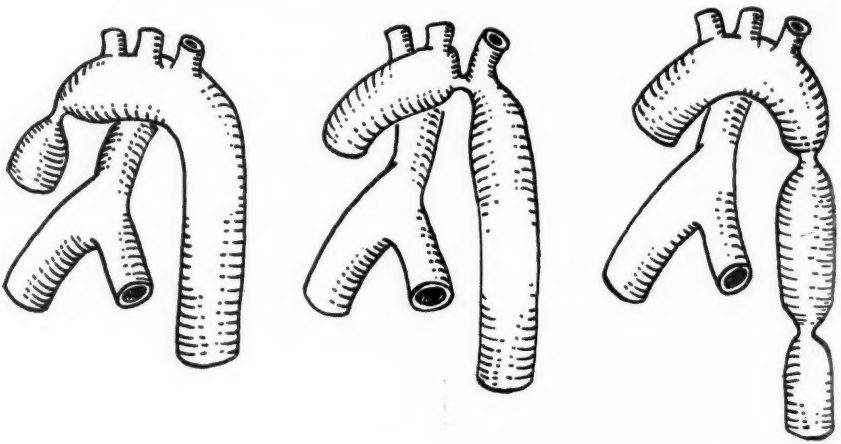
Type V. *Coarctation of the Aorta With Site Elsewhere.*—(Fig. 5.) By definition, this category includes all coarctations located other than at the common site and not specified above. There are only a few such case reports in the literature covering this type, but for the sake of completeness a proper classification must include them. Of all types, this variety is the least frequent.

a. Ascending aorta—Minor degrees of the lesion at this site are compatible with life, but a major degree of involvement may be fatal soon after birth. (Fig. 5, *A.*)

b. Aortic arch—The lesion may occur in this location between the origins of two of the vessels arising from the arch. (Fig. 5, *B.*)

c. Descending aorta—All lesions other than those of hypoplasia are included here that are not Type I, but which occur in the remaining portions of the thoracic or abdominal aorta.

d. Multiple lesions—Various combination of more than one of the above situations may occur. (Fig. 5, *C*.)



A. *B.* *C.*

Fig. 5.—Type V. Coarctation of the aorta with site elsewhere. *A*, Ascending aorta, minor degrees. *B*, Aortic arch. *C*, Multiple lesions.

DISCUSSION

A definite need for a comprehensive yet simple classification of coarctations of the aorta has been attested to in the writings of numerous authors. This need has been magnified by the developments of the current era of surgery. Viewed in the light of its magnitude, the surgical treatment of these anomalies is well defined and established with a very high cure rate and a very low mortality and morbidity rate. It seems self-evident that the cardiovascular surgeons of the world should have a standard, generally accepted, system of designation and classification of these anomalies so that writings, teachings, and communications on the subject will be facilitated.

In the foregoing classification, an attempt has been made to list the lesions by type in the order of their comparative incidence, the most common type being placed first and the rarest last.

It is fitting to explain why the isthmus interruption type (Type IV) with its patent ductus arteriosus should be differentiated from the Type II shunt, distal. The only feature common to these lesions is the patency of the ductus arteriosus producing a right-to-left shunt to the lower aortic segment and venous perfusion of the portion of the arterial bed. Here the similarity ends. If and when the etiology of these anomalies becomes known, we venture to say that the two types under discussion will vary appreciably as to their origin. It

seems to us that the importance of physical absence of a segment of the aorta is of such anatomic, pathologic, and physiologic import as to justify a distinct and separate designation.

The fact that in nearly every Type IV patient either an aortic homograft or a plastic prosthesis will be required to bridge the relatively long defect, whereas simple resection of the coarctation and direct end-to-end anastomosis will suffice in the vast majority of Type II lesions serves further to emphasize their dissimilarity.

SUMMARY AND CONCLUSIONS

The need for a more satisfactory method of classifying coarctations of the aorta has been noted. A brief review of the current confusion and the incompleteness of prevailing methods has been presented.

A completely new and comprehensive, but short and simple system has been devised for their classification and presented with a brief discussion of its propriety.

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BACTERIAL AND MYCOTIC ENDOCARDITIS FOLLOWING CARDIAC SURGERY

Report of a Case

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Robert E. Pence, M.D.
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BACTERIAL endocarditis is now recognized as an occasional complication of cardiac surgery. A review of the literature has revealed a single case of *Candida albicans* endocarditis following cardiac surgery. We report here a case of bacterial and mycotic endocarditis following circumferential suture of the mitral valve ring.

CASE REPORT

A. H., a 32-year-old white male Baptist minister, was admitted to the Cardio-thoracic Surgical Service (Robert P. Glover, M.D., Chief) of The Presbyterian Hospital in Philadelphia on Sept. 1, 1955. His chief complaint was a "rattle on chest" of one year's duration. A heart murmur was noted during an army physical examination in 1942 and, in 1945, he complained of "palpitations." His general condition was good until one year prior to admission when he developed a "rattle on chest," which was relieved in the erect position. He remained active, however, on digitalis and diuretic therapy. He had no peripheral edema and no cough. No history of rheumatic fever could be obtained.

His past medical history included the usual childhood diseases, a tonsillectomy and pneumonia in childhood, and a left nephrectomy at 17 years of age for a "pus kidney." One sister had died of rheumatic fever.

On admission he was a well-developed, well-nourished, white man in no acute distress. The blood pressure was 120/75 mm. Hg and pulse rate 68 per minute. The point of maximal cardiac impulse was felt at the anterior axillary line in the sixth intercostal space. A normal sinus rhythm was present. A Grade 3 "harsh" systolic murmur was transmitted throughout the entire precordium, and a Grade 2 systolic thrill was present at the apex. A Grade 2 "rough" systolic murmur was also present in the aortic area. No diastolic murmurs could be heard. The diagnosis on admission was rheumatic heart disease with predominant mitral insufficiency and associated aortic stenosis. The patient was regarded as a good operative risk.

The results of initial studies were as follows: hemoglobin 15.8 Gm. per 100 ml.; erythrocytes 5,700,000; leukocytes 7,500; normal differential; urinalysis negative; sedimentation rate 0 mm. per hour; bleeding time 1 min., 45 sec.; coagulation time (capillary method) 4 min.; prothrombin time 12.5 sec. (100 per cent). The electrocardiogram was reported as showing combined heart strain and probable atrial hypertrophy. The chest x-rays were reported as compatible with mitral insufficiency and some degree of mitral stenosis.

An attempt at cardiac catheterization was not entirely satisfactory because of venospasm. The right saphenous vein eventually had to be used and the pulmonary artery

From the Laboratories of The Presbyterian Hospital in Philadelphia, Pa.
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could not be entered. The incomplete study revealed a moderate elevation of right ventricular pressure and a normal right atrial pressure. An interventricular septal defect was thereby ruled out.

With a preoperative diagnosis of predominant mitral insufficiency, operation was undertaken on the eighth of September. A total circumferential suture of the mitral ring[1] was accomplished without apparent difficulty. The three-hour operative procedure was well tolerated by the patient. He received 1,500 c.c. of whole blood while on the operating table. At operation, the left atrium and ventricle were moderately enlarged and the right atrium and ventricle were enlarged to a lesser degree. A Grade 4 systolic thrill was felt over the left atrium and pulmonary veins. The aortic valve seemed free of disease after examination by external palpation. No thrombotic material was present in the left auricular appendage. On internal palpation of the mitral valve, a 5-finger annulus with a 3-finger valve orifice was found. The mitral leaflets were thin and pliable but did not approximate in systole. With the purse-string in place the annulus was reduced to a 3-finger diameter with favorable pressure changes. The insufficiency was thought to be improved by 90 per cent.

The immediate postoperative course was uneventful. A few râles in the chest on the fifth day cleared after Thiomerin was given. On the twelfth postoperative day, a chill and fever of 104° F. developed. *E. coli* was recovered from the urine and hemolytic *Staphylococcus aureus* from the blood. A diagnosis of acute bacterial endocarditis was made, and treatment with intravenous penicillin was begun. For eight days the temperature spiked between 99° and 104° F.

From the fifth to the eighth week of hospitalization, the patient's condition remained stable, and the temperature never rose above 98.6° F. Intravenous penicillin was continued for eight weeks, along with other antibiotics (Fig. 1), and then was supplanted by oral penicillin. In less than twenty-four hours, chills and fever of 102° F. recurred, and a hemolytic *Staphylococcus aureus* was again recovered from the blood. Intravenous penicillin was restarted in larger doses, and at the end of the ninth week the patient's temperature was again normal. Some abdominal pain and tenderness, nausea, and tinnitus were present, but disappeared when aspirin was discontinued.

From the tenth to the thirteenth week the patient's temperature remained below 99.4° F., although his general condition remained poor. He appeared weak and lethargic, with occasional bouts of acute pulmonary edema. During the eleventh week, adrenal cortical insufficiency was suspected, and serum electrolyte studies were as follows: Na 121 meq./L., K 5.9 meq./L., Cl 78.8 meq./L., and CO_2 15.7 meq./L. The urinary 17-ketosteroid excretion was 2.3 mg. per twenty-four hours. With hormone therapy, including ACTH, cortisone, and DOCA (desoxycorticosterone acetate), and adequate intravenous electrolyte solutions, the patient's strength and appetite improved.

During the thirteenth week in the hospital, the intravenous penicillin was once more discontinued in favor of oral penicillin. Within thirty-six hours, the patient's temperature began spiking again and varied between 97° and 104.4° F. until death. Hemolytic *Staphylococcus aureus* was once more recovered from the blood during this period. It was suggested that a misplaced "purse-string" might be harboring the organism, in which case the prognosis would seem hopeless. Intravenous penicillin was restarted, but this time without much success. A serum calcium of 8.2 mg. per 100 ml. was recorded following the appearance of physical signs suggesting tetany. Intravenous calcium gluconate relieved this situation and elevated the serum calcium to 10.2 mg. per 100 ml. The patient eventually became extremely lethargic and died 111 days after admission to the hospital.

Autopsy Findings.—At autopsy a diffuse fibrinous pericarditis was found, the pericardium being loosely adherent to the shaggy epicardial surface. The pleurae showed areas of fibrous adhesions, and the right thoracic cavity contained a liter of pale yellow fluid. There was no evidence of arteriosclerosis in the aorta or peripheral vessels. The heart weighed 775 Gm. and showed marked hypertrophy of the myocardium. All cardiac chambers were dilated. The left auricular appendage was absent. There was evidence of healed

rheumatic valvulitis involving mainly the mitral valve, but also the aortic and tricuspid valves. The mitral ring was widely open, admitting 4 fingers easily and measuring 12.0 cm. in circumference. The mitral cusps were markedly fibrosed and contained deposits of calcium. The chordae tendineae were exceedingly thick and firm. The posterior hemicircumference of

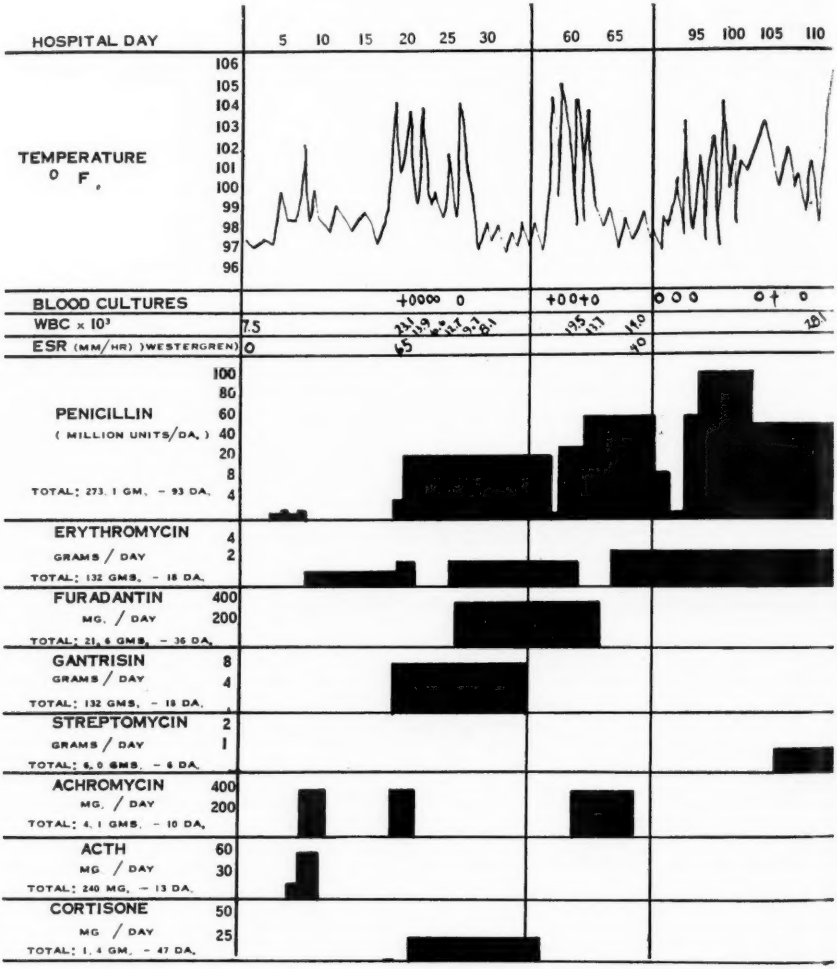

Fig. 1.—Clinical course of patient with deletion of two stable periods.

the "purse-string" suture was seen free in the left auriculoventricular lumen. This portion of the suture measured 6 cm. in length and was covered with a 5 by 2 by 2 cm. irregular, friable, grayish-brown to brownish-red thrombus (Fig. 2). This thrombus was made up of fibrinous material containing colonies of Staphylococci and extensive fungus growth. Shallow

ulcerations covered with fibrinous material were seen at the sites where the suture was attached to the wall of the heart. Microscopic examinations of the heart revealed myocardial hypertrophy, small focal infarcts, focal fibrosis, fat replacement, hemosiderosis, and foreign-body granulomata. Evidence of healed rheumatic valvulitis and reactivated pancarditis was also observed. The areas of ulceration displayed acute and chronic inflammation without bacterial or fungus growth.

The lungs together weighed 1,475 Gm. and showed severe congestion and edema. The spleen weighed 700 Gm. Marked passive congestion and multiple infarcts were noted. A small artery exhibited necrotizing vasculitis, apparently resulting from bacteremia. The

Fig. 2.—View of mitral ring showing intraluminal portion of "purse-string" with thrombus.

left kidney was absent, having been removed previously. The right kidney weighed 400 Gm. and showed compensatory hypertrophy. Several recent infarcts and areas of acute and chronic pyelonephritis were observed, although no cocci or fungi were seen. The liver weighed 2,600 Gm. and showed marked chronic passive congestion. Other findings included moderate depletion of the adrenal cortical lipids, diffuse reactive hyperplasia of the bone marrow, and congestion and slight interstitial inflammation of the pancreas.

DISCUSSION

Fourteen cases of bacterial endocarditis following cardiac surgery have been reported in the literature.[2-8] Thus, while not a common complication, it occurs with sufficient frequency that it must be regarded as one of the hazards of such surgery. In the majority of instances, the offending organism has been a hemolytic staphylococcus.

Mycotic endocarditis is still a rare disease. Kunstadter and associates,[9] in 1952, found 24 cases in the literature and added another due to *Candida albicans*. Several other well-documented reports have appeared in the past five years.[10-1] Recently a case of mycotic endocarditis following cardiac surgery has been reported.[21] Candida appears to be the most frequent offender, having been responsible for endocarditis in 10 out of 25 cases analyzed by Kunstadter and co-workers.[9] Aspergillus,[10-14] Actinomyces,[11, 13, 15] and histoplasmosis[22, 23] have also been reported as the cause of endocardial involvement.

Fig. 3.—Photomicrograph of thrombotic material from "purse-string" showing mycotic growth (periodic acid-Schiff stain).

Endocarditis due to a combination of bacteria and fungi has been previously reported by Joachim,[16] Geiger,[17] Koehlmeier[11] and their associates, but to our knowledge this is the first such report subsequent to surgery of the heart.

At autopsy, a portion of the circumferential suture used to effect reduction in size of the mitral annulus was found lying free in the left auriculoventricular lumen, having been misplaced at the time of surgery. Its presence in the lumen afforded a site for formation of a thrombus in which organisms could lodge and multiply. A hemolytic *Staphylococcus aureus* was recovered from 4 of 17 antemortem blood cultures, and microscopically the thrombotic material from the

"purse-string" contained colonies of Staphylococci. In addition, this thrombotic material also showed extensive mycotic growth. The fungi, identified as *Candida albicans* with hematoxylin and eosin and periodic acid-Shiff stains,* were arranged in a foliate pattern, showing septate mycelia and yeastlike cells (Fig. 3). Since the mycotic component was entirely unsuspected at the time of autopsy, culture studies were not done.

The extensive use of antibiotics and chemotherapeutic agents in recent years apparently has resulted in an increased incidence of mycotic infection,[9, 12, 18, 19] as predicted by Zimmerman in 1950.[14] Evidence has also been presented that widespread use of steroid hormones may be responsible for enhancement of fungous infections.[18, 19, 20] In addition, increased susceptibility due to the primary disease and the nature of the local lesion is also important.[10, 19] In the case presented all of these conditions probably obtained in favoring growth of the fungus.

While differences between sensitivity of organisms to antibiotics in vitro and in vivo are well recognized, it is of interest here that this Staphylococcus was penicillin resistant in vitro, yet seemed to be controlled by large doses of intravenous penicillin. Although other antibiotics were used sporadically, chills and fever with positive blood cultures promptly recurred on the two occasions when intravenous penicillin was discontinued in favor of smaller doses of oral penicillin.

SUMMARY

A case of bacterial and mycotic endocarditis following circumferential suture of the mitral ring is reported. Its unusual features and the probable contributory factors are discussed.

We are indebted to Dr. Robert P. Glover and his associates for permission to use this material and to Mr. Joseph Sunner for the illustrations and photograph.

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EXPERIMENTAL BILATERAL TRANSSTERNAL THORACOTOMY: FACTORS IMPROVING SURVIVAL

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INTRODUCTION

A BILATERAL transsternal thoracotomy affords optimal access to the heart and great vessels, and is of particular value in experimental surgery because it simulates the technical exposure employed in human patients during most intracardiac and pericardiac operations. Earlier, this approach was attended by a high mortality in our laboratory, and many visitors to our department stated that they had abandoned this incision in their own experimental preparations in favor of the less traumatic unilateral thoracotomy, because of inability to obtain long-term survival following the more extensive procedure.

In conjunction with our recent efforts to prevent ventricular fibrillation during right ventriculotomy and hypothermia, an experimental operative technique which reproduced its clinical counterpart was considered essential. This required the bilateral transsternal exposure, and stimulated us to evolve a program of operative management and postoperative care which would yield an acceptable mortality rate.

MATERIALS AND METHODS

Mongrel dogs weighing from 6.4 to 16.3 Kg. were chosen at random from a large isolation run. The only selection involved was an attempt to exclude those animals which were obviously ill or within the extremes of age. None received any special preoperative diet or therapy. No pre-anesthetic medication was employed. Sodium pentobarbital was administered intravenously as the anesthetic. The dosage was adjusted to eliminate the lateral canthal reflex with preservation of the medial, which required about 30 mg. per kilogram of body weight. Following tracheal intubation, the animals were placed in an ice-water bath, and positive-pressure respiration was begun with 100 per cent oxygen, at about 40 respirations per minute. Removal from the ice water was done when a rectal temperature of 29° C. was reached. A further decline of 3° to 5° C. occurred, so that the experimental procedures were performed at a rectal temperature of 24° to 26° C.

From the Halsted Experimental Laboratory of Surgical Research, Department of Surgery, University of Colorado, Denver.
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All of the animals underwent bilateral transsternal thoracotomy through the fourth intercostal space. Right ventriculotomy was then performed during an inflow-outflow circulatory occlusion of from 8.5 to 13.0 minutes.

After the release of the inflow occlusion and reconstitution of a normal circulation, the following protocol was observed carefully:

1. Continued observance of aseptic technique.

2. Loose closure of the pericardium with interrupted sutures of No. 4-0 black silk.

3. Bilateral water-sealed chest suction for 2 to 8 hours, depending on the immediate postoperative activity of the animal.

4. Approximation of the sternum with two crossed parasternal No. 28 or No. 32 wires.

5. Approximation of the rib cage on either side with a single No. 28 or No. 32 paracostal wire.

6. Careful four-layer (intercostal bundle, pectoral muscles, fascia, and skin) closure of the thorax.

7. Replacement of blood loss with whole blood or a plasma-volume expander—about 10 to 15 ml./Kg.

8. Intramuscular administration of Metrazol (pentylenetetrazol) 100 mg. (1 ampule) per kilogram of body weight, immediately postoperative.

9. Rewarming in a water bath at 40° to 45° C., where the animal remained until a rectal temperature of 32° to 36° C. was achieved (spontaneous respiration always followed cessation of the positive-pressure breathing in this temperature range).

10. Routine bronchoscopy on the morning following operation of all dogs weighing 10 Kg. or less, and immediate bronchoscopy of all other dogs developing persistent cough on the first or second postoperative day, small doses of sodium thiopental being used intravenously as the anesthetic.

11. Administration of antibiotics (penicillin and streptomycin) for the first 72 postoperative hours.

If ventricular fibrillation occurred at any point during the operative procedure, it was allowed to persist throughout the predetermined period of circulatory occlusion. Then electrical ventricular defibrillation was achieved as described elsewhere, with particular attention to the prevention of epicardial and myocardial burns.[1]

"Survival" has been assigned arbitrarily as implying "alive and well" seven days postoperative.

RESULTS

Thirty successive dogs underwent bilateral transsternal thoracotomy, hypothermia, and right ventriculotomy as described. Of these, 10 developed ventricular fibrillation during the operative procedure, and each was successfully defibrillated electrically. Twenty-nine animals survived. The one death occurred as a result of an overdose of barbiturate prior to bronchoscopy on the first postoperative day (Table I).

TABLE I. SURVIVAL OF DOGS UNDERGOING BILATERAL TRANSSTERNAL THORACOTOMY DURING HYPOTHERMIA

DOGS UNDERGOING TRANSSTERNAL THORACOTOMY	ALIVE 7 DAYS POSTOPERATIVE	SURVIVAL
30	29*	97%

*The one death was due to an overdose of barbiturate prior to bronchoscopy.

DISCUSSION

The protocol of thoracic closure and early postoperative care as presented here permits the use of the bilateral transsternal thoracotomy in experimental animals without imposing an increased operative hazard. The relative importance of each component has not been evaluated, but presumably no single aspect is crucial.

Closure of the pericardium assures a stable midline position of the heart; otherwise, torsion of the venae cavae could occur with resultant embarrassment of right heart filling when the animal is turned from side to side during the recovery period.

Careful examination of the thorax, followed by underwater drainage of both hemithoraces, permits complete and permanent expansion of the lungs. Meticulous suture of the posterior angles of the incision as well as the parasternal regions is essential.

Maintenance of a normal blood volume appears as practical here as in other major operative procedures.

Rapid rewarming and reversal of barbiturate effect with Metrazol allows the animal to be awake and ambulatory a few hours following operation. In addition, Metrazol encourages deep breathing with its attendant benefits. No convulsions have been noted following the use of this analeptic in the dosage employed here. No clinical evidence of heart failure (tachycardia, râles, or distention of tongue veins) was observed during or after the period of rapid rewarming to a rectal temperature of 33° to 38° C.

On the morning following operation, bronchoscopy was performed in most animals. In those that underwent endoscopy, mucous plugs of varying sizes were recovered from the tracheobroncial tree. A complete cast of the lower trachea and both bronchial trees was removed from one animal. The formation of these accumulations of mucus is thought to be secondary to poor tussive efforts in the early postoperative period, and serves as a further recommendation for the expeditious return of the animal to consciousness postoperatively.

In addition to the technical advantage of the transsternal thoracotomy, a more subtle indication for its use in cardiovascular research may exist.

Hypothermia has proved to offer a satisfactory means of correcting certain cardiovascular anomalies under direct vision, i.e., interatrial septal defect, pulmonary stenosis, and aortic stenosis.[2-5] However, operating within the open ventricles during hypothermia has had limited application because of the threat of ventricular fibrillation. Recently, efforts have been made in many laboratories to overcome this disadvantage.[6-10] However, the basic experi-

mental preparation varies from one investigating group to another. Our own experience in the past two years suggests that the bilateral thoracotomy is associated with a higher incidence of ventricular fibrillation during hypothermia than is the unilateral approach. If this is indeed so, standardization of this fundamental aspect of the experimental preparation among research groups would seem advisable.

The standard procedure of choice may be the bilateral approach, since it duplicates the clinical situation under which many of the experimental techniques will eventually stand trial.

SUMMARY AND CONCLUSIONS

Bilateral transsternal thoracotomy provides optimal exposure of the heart and great vessels. It is of particular value in experimental cardiac surgery, because it duplicates the clinical situation and provides a standard preparation for interlaboratory comparisons. A method of wound closure and early postoperative management is presented which permits performance of this more extensive procedure with little or no increase in operative risk.

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PNEUMOMEDIASTINUM: PHYSIOLOGIC OBSERVATIONS

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AIR may enter the mediastinum following trauma involving the trachea or esophagus, during operative procedures in the neck in which the pretracheal fascia has been opened, or "spontaneously" from the lung. Forbes and co-workers[1] have demonstrated experimentally that, following opening of the pretracheal fascia as in a tracheotomy, air may be sucked into the mediastinum to produce mediastinal emphysema and a pneumothorax. This process is augmented by labored respiration. Bouden and Schweizer,[2] in 1950, reported 18 cases of pneumothorax and 2 cases of mediastinal emphysema following operations upon the head and neck, which developed presumably by this same mechanism.

That these patients have severe symptoms, even leading to death, has been reported and confirmed many times. Usually it has been assumed that the mechanism producing the symptoms is the development of air under tension in the mediastinum. This causes embarrassment of the venous return in the venae cavae, lowered cardiac output with hypotension, and circulatory failure unless the pressure is released. As there is some reason to doubt that sufficient pressure can be developed in the mediastinum to impede venous return seriously, this problem has been experimentally reappraised.

Jehn and Nissen[3] injected air into the mediastina of puppies and did produce asphyxia with a falling arterial blood pressure leading to peripheral circulatory collapse. However, these experiments were not controlled to determine the effects of the frequently accompanying pneumothorax. Later, Ballon and Francis[4] injected air into the mediastinum, inflated rubber balloons in the posterior mediastinum, or cut windows in the lower trachea to produce mediastinal emphysema. Here again, these studies were not adequately controlled to determine the effects of frequently accompanying pneumothorax. Nor does a rubber balloon simulate free air in the mediastinum as the confines of the mediastinum are not as definite as those of the rubber balloon.

METHODS

Rabbits and adult mongrel dogs were anesthetized with pentobarbital, and a long needle was introduced through the jugulum passing through the trachea into the visceral space of the upper mediastinum. Air was injected slowly

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in graded amounts with constant recording of the pressure in the mediastinal air pocket. Intercostal catheters were inserted bilaterally and attached to water-seal drainage with suction to detect the development of a pneumothorax. Catheters were likewise introduced into the venae cavae, the right atrium, and the femoral artery. Pressures were recorded by mercury manometers and on Sanborn electromanometers using Statham strain gauges.

Four dogs had poudrage of both pleural cavities with 5 per cent iodized talc to produce a sterile pleuritis and complete pleural symphysis. After a period of three to four weeks, these dogs were subjected to the experiment as outlined above to check if significant tension could be produced when the lateral mediastinal fascias were buttressed by the lung.

Rabbits were used in addition to dogs because of the concept that the dog has an instable or flimsy mediastinum. Our anatomic studies demonstrated that, while the anterior mediastinum of the dog is composed only of a sheet of fatty mesentery and is quite mobile and tenuous, the mediastinal fascias surrounding the visceral space are quite tough and have the same anatomic features as that of other experimental animals. In fact, it appears to us that these fascias in the dog are tougher than those of the rabbit. Both experimental animals differ from human beings in that the exposed inferior vena cava projects through the right pleural space covered only by a thin sheet of pleura and completely surrounded by lung while, in man, the inferior cava has relatively a much shorter course in the thorax before entering the right auricle and is much better protected from pleural pressures.

Last, to ascertain whether the experimental results on these animals might be validly transferred to man, the rupturing pressure of the human mediastinum was measured at the autopsy table. After removal of the breast plate, the chest was flooded with water. A long needle was injected through the jugulum into the visceral space of the posterior mediastinum, and air was injected as in the experimental animals with constant pressure recordings.

RESULTS

Our method was universally successful in producing marked mediastinal emphysema in all animals. It was found that the rapid injection of air under high pressure led immediately to a pneumothorax. Accordingly, initial injections of air were made very slowly with constant monitoring to prevent a rise of the pressure above 40 mm. Hg.

In only 1 of 16 normal dogs was any marked effect produced by the pneumomediastinum alone. Dog 10 developed very extensive mediastinal emphysema, subcutaneous emphysema, and an extremely tense pocket of air in the cervical mediastinal visceral space which could be easily seen and felt. During this period, however, while the superior vena caval pressure rose 4 mm. Hg (equivalent to approximately 52 mm. of water pressure), the right atrial pressure showed no change, and the femoral arterial pressure fell only 24 mm. Hg. The inferior caval pressure showed no change. Cervical mediastinotomy on this dog immediately relieved these slight pressure changes and,

in addition, helped to decompress the accompanying right pneumothorax. Further air injected into the mediastinum left via the mediastinotomy, preventing further complications from the pneumothorax.

In the remaining 15 dogs, all observable effects were due solely to the accompanying pneumothorax (Fig. 1). This invariably effectively decompressed the mediastinal emphysema but in itself proved fatal if unrelieved. The effects

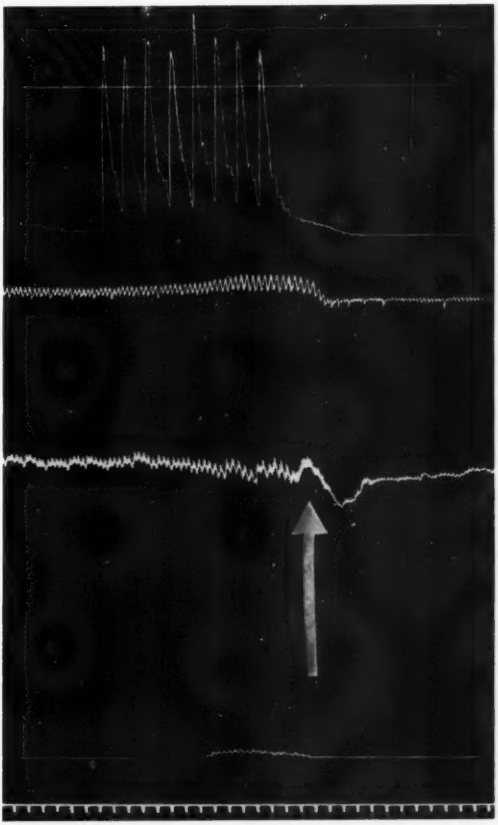

Fig. 1.—Pneumothorax effect. *Top line* = base line. High spiked line reflects the pressures in the mediastinal air pocket, showing the immediate fall of pressure at the conclusion of each 50 c.c. injection. *Next line* = right atrial pressure. *Fourth line* = femoral arterial pressure, and *lower line* shows the inferior vena caval pressure. Time markers represent 5 seconds. At the arrow, the tension pneumothorax was decompressed. Note the return of all measurements to normal.

of the tension pneumothorax were easily reversed by aspiration unless there had been too much delay. The typical preterminal response was a marked rise in the arterial, atrial, and vena caval pressures, followed by a rapid decline and death (Fig. 2). This is a characteristic response to progressive acute anoxia, regardless of the cause.

In addition to rupturing into the pleural cavities, air was found to dissect into the subcutaneous tissues or inferiorly along the fascial planes into the retroperitoneal spaces (Table 1). Usually one of these routes predominated.

TABLE I. SITES OF AIR-DISSECTION

TOTAL	C.A.P.	P.T.		R.P.	S.E.
		RT.	LT.		
16 (Dogs)	5	13	12	1	5
4 (Poudraged Dogs)	4	3	2	4	1
5 (Rabbits)	0	4	4	1	5

Sites of air-dissection noted in experimental animals. C.A.P. = cervical air pocket; P.T. = pneumothorax; R.P. = retroperitoneal; S.E. = subcutaneous emphysema.

Frequently, subcutaneous emphysema was noted first and, once this route of escape was established, several liters of air could be injected rapidly into the posterior mediastinum with no deleterious effects being observed.

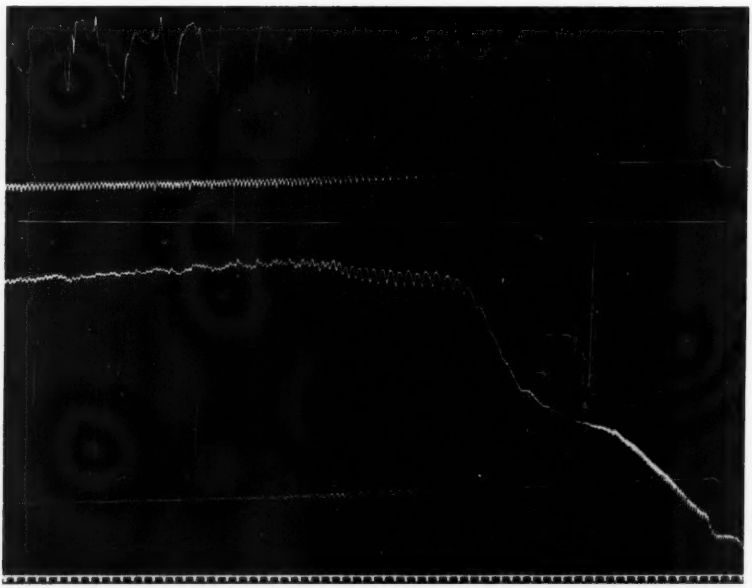

Fig. 2.—Terminal anoxic effect of tension pneumothorax. Tracings are the same as in Fig. 1, except for shift of the base line. Note the rising caval and atrial pressures and the initial rise of the arterial pressure with its terminal fall. Again the mediastinal air pocket pressure is seen to return to normal after each injection.

Similarly, marked mediastinal emphysema was produced in 5 rabbits. No rabbit developed physiologic effects from the mediastinal emphysema alone. The rabbits were much more prone to develop subcutaneous emphysema than the dogs. They likewise developed unilateral or bilateral tension pneumothoraces and showed only the effects of the tension pneumothoraces. Retroperitoneal air was noted in only one of the rabbits.

In the 4 poudraged dogs, only a partial pleural symphysis was obtained. Extensive mediastinal adhesions, however, were noted in each, and the mediastinal pleura appeared to have been effectively strengthened, for these animals developed pneumothoraces late and only after the appearance of extensive subcutaneous and retroperitoneal collections of air. All developed a very tense cervical air pocket within the visceral space.

Effects from mediastinal emphysema alone were noted in 3 of these 4 dogs (Table II). These changes, while definite, did not cause any appreciable circulatory distress and all were transient. Respiratory difficulty was never occasioned by the pneumomediastinum, subcutaneous emphysema, or retroperitoneal air—only by the pneumothorax.

TABLE II. POUDRAGED DOGS—PRESSURE CHANGES (mm. Hg)

DOG	V.C.	R.A.	F.A.	C.A.P.
1	+6	+4	−40	+6
2	+2	0	0	+4
3	0	0	−10	+4
4	0	0	0	+5

Pressure changes noted in poudraged dogs. V.C. = vena cava; R.A. = right atrium; F.A. = femoral artery; C.A.P. = pressure in cervical air pocket of the visceral space.

The deep cervical air pockets could quickly be decompressed by a cervical incision extending through the pretracheal fascia which is the anterior encompassing fascia of this pocket. Similarly, mediastinotomy would prevent a pneumothorax in most cases, but not always, for some dogs were observed to develop a tension pneumothorax even in the presence of an effective mediastinotomy which was bubbling air freely. Dog 13 was noted to have both an extremely tense cervical air pocket in the mediastinum and a right pneumothorax. The attendant severe respiratory and circulatory symptoms were not relieved by mediastinotomy but by decompression of the pneumothorax. Only rarely could the pneumothorax effect be reversed by mediastinotomy.

In the post-mortem observations on 6 human adults, air was injected slowly to keep the pressure below 20 mm. Hg. Again, the pressure immediately dissipated as air dissected diffusely through the mediastinum. In no case could a persistent tension above 5 mm. Hg be maintained. Air always bubbled out into one or both pleural cavities after injection of only a small amount of air —varying from 20 to 400 c.c. The point of rupture was usually anterosuperior, far from the site of injection.

DISCUSSION

Air injected into the mediastina of rabbits, dogs, and human beings does not produce a tension effect as the air escapes by dissecting along the tissue planes or rupturing into the pleural spaces, and the mediastinal pressure quickly falls to insignificant levels. At no time were we able to develop a persistent pressure within the normal mediastinum greater than about 5 mm. Hg. In dogs which have had the mediastinal spaces buttressed by formation of adhesions or symphysis of the mediastinal and visceral pleurae, slightly greater pressures can be developed which may produce mild circulatory symptoms.

Goldberg, Mitchell, and Augrist[5] found that the rupturing pressure of the mediastinum in infant human cadavers is approximately 40 mm. Hg. At this level, the air would rupture into the pleural space. A slight shift in position might close this rent and often a subsequent pneumothorax would appear on the opposite side.

Our results on human adults would suggest that such a pressure can be attained only with the acute injection as the dissection of the air along tissue planes immediately reduced the pressure to less than 5 mm. Hg. In addition, in each observation the mediastinum ruptured with egress of air into the pleural cavity at pressures less than 20 mm. Hg. It is probable that, in the presence of pulmonary disease with pleural thickening, much higher acute pressures may be attained before the pleura ruptures. Nonetheless, the air will dissect subcutaneously and retroperitoneally and, only in most unusual circumstances, develop a persistent high pressure within the confines of the mediastinum.

This work suggests that symptoms due to a tense pneumomediastinum must be rare and that the effects noted are due to an associated pneumothorax. In fact, once a pneumothorax has developed, it appears to be quite impossible to develop sufficient pressure within the mediastinum to cause any degree of compression of the heart or great vessels. The rise in right atrial pressure noted in all dogs with pneumothoraces suggests that the block is in the pulmonary capillaries and not the venae cavae.

Many of the clinical reports such as those of Karns,[6] Sheinfeld,[7] Gaudrault,[8] and Rydell[9] attribute the beneficent effects of emergency cervical mediastinotomies to decompression of massive mediastinal emphysema. They do not present adequate supporting data, however, to show that the symptoms were due to the mediastinal air. In fact, most of the patients presented had accompanying pneumothoraces. Our experimental observations support the contention of Tuttle[10] that the difficulty is usually due to the tension pneumothorax and not the mediastinal emphysema.

The mediastinal emphysema that we have studied is, of course, completely different from malignant interstitial emphysema of the lungs accompanied by mediastinal emphysema. This has been extensively studied by the Macklins,[11] Hamman,[12] and more recently by Fagin and Schwab.[13] In this condition, the air dissects from the alveoli along vascular sheaths and compresses the pulmonary capillaries. It may, in addition, cause air lock of the alveoli, preventing adequate ventilation. Vanderlaan and Maresh[14] have reported one such case of mediastinal emphysema without a pneumothorax. The patient developed subcutaneous emphysema, cyanosis, dyspnea, and pain and became irrational. A collar incision allowed an immediate egress of air with rapid improvement and an uneventful convalescence. This type of case apparently is extremely unusual.

A mediastinotomy can be of value in preventing a pneumothorax or in preventing further accumulation of air in an existing pneumothorax. It is doubtful, however, that the mediastinotomy will decompress an existing pneumothorax.

In addition, it should be stressed that when a cervical mediastinotomy is performed for mediastinal emphysema, the pretracheal fascia which forms the anterior sheath of the visceral space in the neck must be opened to decompress the mediastinum effectively. A collar incision extending only into the subcutaneous tissues cannot be effective.

SUMMARY

1. Experimental mediastinal emphysema produced tension effects in only 1 of 16 dogs and in none of 5 rabbits. The only significant circulatory and respiratory effects were due to secondary tension pneumothoraces.

2. After poudrage of the pleural spaces of 4 dogs, slightly more impressive circulatory changes were noted in 3.

3. The pneumomediastina in all are immediately decompressed by the air dissecting into the subcutaneous tissues or along fascial planes into the retroperitoneal space, or rupturing into the pleural cavities.

4. The rupturing pressure of the mediastinum of the normal adult human was found to be less than 20 mm. Hg. Due to dissection of the air along the fascial planes, the mediastinal pressure could never be maintained even as high as 5 mm.

5. Cervical mediastinotomy may be of value in the occasional case of mediastinal emphysema, but is of value most frequently in preventing further accumulations of air in the accompanying pneumothorax.

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UNILATERAL PULMONARY ARTERY OCCLUSION IN MAN

II. Studies in Patients With Chronic Pulmonary Disease

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INTRODUCTION

The technique of unilateral pulmonary artery occlusion has been used in our laboratory in over 100 cases to date. The results in a group of controls have already been reported, with particular reference to flow-pressure relationships.[1, 2] It has been demonstrated that this technique provides a method for increasing unilateral pulmonary blood flow without increasing cardiac output as, for example, by exercise.

It is the purpose of this communication to present the results of studies carried out in a series of patients with various types of chronic pulmonary disease, utilizing the method of temporary unilateral pulmonary artery occlusion.

Standard pulmonary function tests and cardiac catheterization, as well as arterial blood studies, have contributed much to the understanding of the pathophysiology of such diseases.[3, 4] There are still certain problems, however, which remain unsettled. Of particular importance are those patients being considered for resectional lung surgery who are poor risks. In such cases, impaired ventilatory function together with elevated pulmonary arterial pressure has usually been considered a contraindication to such surgery, and the decision to proceed is still empirical.

Recently described work, by Perkins and co-workers,[5] on ventilation-perfusion relationships in the lung and, by Riley and associates,[6-8] on maximal diffusion capacities, promises to aid greatly in evaluating patients with chronic pulmonary disease. Certain results obtained in the present study extend the results by these authors. The clinical application of the method of unilateral occlusion in the study of these parameters in patients with various forms of chronic pulmonary disease is herein presented.

METHODS AND MATERIAL

Cardiac catheterization was carried out in the usual manner using light sedation (usually secobarbital, 0.1 Gm.). Pulmonary artery pressures were recorded using a Sanborn electromanometer and a direct writing four-channel Sanborn recorder. Peripheral arterial pressures were recorded via Statham strain gauge manometers. Simultaneous mixed venous (pulmonary artery) and systemic arterial (femoral or brachial) blood samples were analyzed for oxygen content by the Van Slyke method[24] and the spectrophotometric method of

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Hickam and Frazer.[25] Oxygen consumption and minute ventilation were determined on a Collins spirometer in the earlier studies and, more recently, by collection in a Douglas bag and analysis on a Pauling oxygen analyzer. Cardiac

TABLE I. PHYSICAL CHARACTERISTICS AND DIAGNOSIS OF PATIENTS WITH CHRONIC PULMONARY DISEASE

CASE	AGE	SEX	S.A.	V.C.	M.B.C.	EXP. 1 SEC.	DIAGNOSIS
A. *Emphysema (Simple)*							
1. J. B.	32	M	1.80	4.0 (82%)	123	47.0%	Localized emphysema
2. G. F.	53	M	1.86	3.7 (80%)	72.2 (66%)	41.8%	Chronic emphysema
3. V. M.	56	M	1.47	2.2 (61%)	39.2 (46%)	39.7%	Chronic emphysema, cor pulmonale
4. G. H.	48	M	1.47	3.1 (83%)	35.1 (38%)	63.0%	Chronic emphysema
5. M. H.	40	M	1.88	2.6 (55%)	34.0 (28%)	38.5%	Chronic emphysema, cor pulmonale
6. H. W.	61	M	1.68	2.0 (47%)	23.7 (25%)	35.4%	Chronic emphysema
7. B. S.	57	M	1.86	1.9 (41%)		25.0%	Chronic emphysema, cor pulmonale
8. M. H.	55	F	1.98	2.3 (59%)	48.1 (53%)	50.0%	Chronic emphysema, cor pulmonale
B. *Emphysema (Complicated)*							
9. L. G.	68	M	1.54	1.6 (42%)	28.8 (36%)	42.0%	Bullous emphysema
10. L. D.	51	M	1.69	1.6 (38%)	39.0 (38%)	74.3%	Bullous emphysema
11. R. H.	42	M	1.87	2.2 (47%)	46.3 (38%)	32.5%	Cystic disease
12. M. H.	40	M	1.50	1.7 (47%)	37.2 (37%)	53.0%	Cystic bronchiectasis
C. *Granulomatous Lung Disease*							
13. V. S.	23	F	1.60	3.2 (80%)	106 (108%)	48.5%	Eosinophilic granuloma
14. D. D.	36	F	1.52	2.0 (66%)	63.7 (77%)	47.5%	Sarcoidosis
15. W. T.	54	M	1.69	2.8 (65%)	37.9 (38%)	34.2%	Silicosis
16. W. B.	47	M	1.79	2.1 (50%)	55 (53%)	50.7%	Granulomatous disease; etiology undetermined
17. C. E.	52	M	1.99	3.0 (60%)	57 (50%)	Total 7 sec.	Cavitary tuberculosis
18. B. L.	73	M	1.58	2.4 (60%)	56.6 (74%)	70.0%	Multiple pulmonary emboli
D. *Carcinoma of Lung*							
19. F. B.	63	M	1.64	2.5 (61%)	78.0 (89%)	55.5%	Primary bronchiogenic carcinoma
20. F. M.	68	M	1.41				Primary bronchiogenic carcinoma
21. G. W.	51	M	1.54	2.0 (51%)	61 (67%)	65.0%	Primary bronchiogenic carcinoma
22. F. B.	49	M	1.67				Primary bronchiogenic carcinoma
E. *Pulmonary Disease With L.V. Failure or Mitral Disease*							
23. E. W.	53	M	1.57				Emphysema, rheumatic heart disease with mitral insufficiency
24. O. B.	66	F	1.57				Periarteritis, tuberculosis
25. E. O.	25	F	1.51	2.1 (70%)	85.0 (94%)	74.0%	Pulmonary and myocardial sarcoid with left ventricular failure
26. R. H.	43	M	1.72	3.7 (86%)	49.8 (45%)	40.0%	Emphysema, rheumatic heart disease with mitral insufficiency
27. W. B.	48	M	2.12	4.2 (80%)	75.0 (54%)	53.7%	Emphysema, bronchiectasis, rheumatic heart disease with mitral insufficiency
28. D. E.	58	M	1.96	3.8 (61%)	57.8 (52%)	44.5%	Emphysema, arteriosclerotic heart disease with mitral insufficiency

S.A. = surface area; V.C. = vital capacity; M.B.C. = maximum breathing capacity; Exp. 1 Sec. = per cent of vital capacity expired in first second.

TABLE II. CATHETERIZATION DATA IN PATIENTS WITH CHRONIC PULMONARY DISEASE

CASE NO.		ARTERIAL PRESSURES: PULMONARY S	PULMONARY D	PULMONARY M	BRACHIAL S	BRACHIAL D	BRACHIAL M	BLOOD OXYGEN CONTENT: CAP.	ART.	% SAT.	V	A-V	O_2	CO	CI
1. J. B.	C	25	7	15	135	75	92	17.5	16.0	91	12.1	3.9	252	6.5	3.6
	C_o	25	7	15	140	80	98	17.5	17.3	99	13.0	4.3	252	5.9	3.2
	O_o(e)	32	10	20	140	85	100	17.5	17.7	100	13.2	4.5	252	5.6	3.1
	O_oE	41	15	27	145	100							468		
2. G. F.	C	20	5	10	100	65	80	19.8	18.2	92	14.0	4.2	434	10.3	5.6
	C_o	20	5	10				19.8	19.6	99	15.3	4.3	434	10.1	5.5
	O(e)	30	8	15				19.8	18.5	94	14.3	4.2	396	9.4	5.1
	O_o	25	5	13				19.8	19.4	98	13.7	4.7	396	8.4	4.5
3. V. M.	C	42	10	18				17.6	13.3	76	8.9	4.4	168	3.7	2.5
	C_o	38	12	22				17.6	17.2	97	12.7	4.5	168	3.7	2.5
	O(p)	61	18	32				17.6	14.7	84	9.2	5.5	174	3.2	2.2
	O_o	56	10	26				17.6	17.3	98	12.9	4.4	174	4.0	2.7
4. G. H.	C	30	18	25	96	60	72	22.4	19.6	88	13.3	6.3	361	5.7	4.0
	C_o	42	12	22				22.4	22.4	100	16.4	6.0	361	6.0	4.2
	O(e)	59	35	40	130	95		22.4	18.7	84	11.7	7.0		5.2	3.6
5. M. H.	C	48	18	33				17.1	13.4	79	7.9	5.5	460	8.4	4.4
	C_o	43	18	30				17.1	15.0	88	9.6	5.4	460	8.5	4.4
	O_o(e)	62	25	40				17.1	13.7	80	6.7	7.0	716	10.2	5.4
6. H. W.	C	55	5	25				19.8	15.5	78	12.8	2.7	400	14.8	8.6
	C_o	42	13	22				19.8	19.5	98	14.5	5.0	400	8.0	4.6
	O(p)	65	16	40				19.8	19.1	96	14.7	4.4	470	10.7	6.2
7. B. S. (a)	C	65	30	45	125	60	80	18.2	12.2	67	5.3	6.9	170	2.5	1.4
	C_o	65	30	45	125	75	82	18.2	17.1	94	11.8	5.3		3.2	1.9
	O(e)	66	30	45	125	75	82	18.2	13.2	73	6.3	6.9		2.5	1.4
	O_o							18.2	17.7	98					
B. S. (b)	C	54	30	40	100	70	78	18.4	14.8	81	9.6	5.2	150	2.9	1.7
	CE	60	30	44				18.4	15.3	83	8.8	6.5	200	3.1	1.8
	O(e)	58	28	40	125	75	80	18.4	14.9	81	9.2	5.7	150	2.6	1.5
	O_o	54	30	40				18.4	17.6	96	12.0	5.6	150	2.7	1.6
8. M. H.	C	82	28	40				19.8	15.5	78	11.7	3.8	300	7.9	3.9
	C_o	72	22	38				19.8	19.5	98			(Assumed)		
	O(p)	92	30	46				19.8	15.2	77	12.1	3.1	300	9.7	4.8
	O_o	84	20	42				19.8	19.3	97	14.0	5.3	300	5.7	2.8

9. L. G.	C	44	14	26	100	30	50	20.2	15.6	77	12.0	3.6	289	8.0	5.3
	C_o	50	18	28				20.2	19.8	98	15.4	4.4	307	7.0	4.7
	O(p)	75	25	42	130	65	88	20.2	15.6	77	11.5	4.1	307	7.5	5.0
	O_o	62	20	34				20.2	19.9	99	15.9	4.0	289	7.2	4.8
10. L. D. (a)	C	48	27	37	130	80	100	19.1	15.4	83	10.4	5.5	253	4.6	2.7
	C_o	43	26					19.1	18.4	96	13.9	4.5		5.6	3.3
	O_o(c)	70	22	42				19.1							
L. D. (b)	CRPA	40	20	30											
	ORPA	70	35	50	150	90	120	20.2	16.7	83	11.4	5.3		4.0	2.7
	CLPA	40	20	30	150	90	120	20.2	18.2	90	11.7	6.5		3.2	2.1
	OLPA	60	30	42	160	100	125	20.2	17.4	86	11.7	5.7	215	3.8	2.5
	$OLPA_o$	50	25	35	175	100	125	20.2	20.0	99			210		
11. R. H.	C	45	25	33	100	75		20.1	16.4	82	13.6	2.8	360	12.8	7.2
	C_o	32	18	24				20.1	19.5	97	15.1	4.4		8.2	4.6
	O(p)	68	24	45				20.1	16.7	84	13.0	3.7	400	10.8	6.1
	O_o	69	22	42				20.1	19.4	97	15.3	4.1		9.8	5.5
	O_oE	78	28	56											
12. M. H.	C	54	31	40	150	77	105	17.6	16.0	91	11.2	4.8	188	3.9	2.5
	C_o	58	18	32	135	68	90	17.6	17.6	100	13.1	4.5	188	4.2	2.7
	O(p)	70	20	37	140	65	90	17.6	15.9	91	11.6	4.3	186	4.3	2.7
	O_o	64	15	35	135	68	90	17.6	17.6	100	13.0	4.6	186	4.1	2.6
13. V. S.	C	15	5	10								5.6	140		2.5
	O(c)	30	10	20								(Assumed)			(Assumed)
14. D. D.	C	24	9	15	105	75	88	16.1	14.3	89	9.4	4.9	239	4.9	3.2
	C_o	28	10	15				16.1							
	O_o(p)	44	20	30	105	75	88	16.1	15.8	98	11.2	4.6	271	5.9	3.8
	O_oE	60	20	35				16.1	16.1	100	10.0	6.1	453	7.4	4.8
15. W. T.	C	30	19	22	100	55	75	21.2	19.1	90	14.6	4.5	161	3.6	2.1
	C_o	30	15	20	105	70	80	21.2	21.0	99	15.6	5.4		3.0	1.7
	O_o(p)	58	36	38	125	75	100	21.2	19.0	90	13.6	5.4	214	4.0	2.3
	O_o	52	22	34	105	65	80	21.2	20.4	99	15.2	5.7		3.8	2.1
	O_oE	70	40	55	140	90	115	21.2	19.1	90	11.2	7.9	535	6.8	3.9
	CE	42	18												

C = control (unoccluded); O = unilateral pulmonary artery occlusion; E = exercise; C_o on Collins spirometer, breathing 100 per cent oxygen; O_o = occluded, on oxygen; O_oE = exercise while occluded, on oxygen; S = systolic; D = diastolic; M = means; Cap. = blood O_2 capacity; Art. = arterial oxygen content; % Sat. = per cent saturation; V = mixed venous (pulmonary artery) oxygen content; A-V = arteriovenous oxygen different; O_2 = oxygen consumption in cubic centimeters per minute; CO = cardiac output; CI = cardiac index; pressures are in mm. Hg, oxygen contents in volumes per cent.

Where indicated following O (occlusion) (p) indicates occlusion of part of the pulmonary blood flow to the right lung, either right lower lobe or right lower and middle lobes. (c) indicates occlusion of the right main pulmonary artery. In Case 10b first the right main pulmonary artery was occluded, indicated by RPA, and then the left, indicated by LPA.

TABLE II—CONT'D

CASE NO.		ARTERIAL PRESSURES						BLOOD OXYGEN CONTENT							
		PULMONARY			BRACHIAL										
		S	D	M	S	D	M	CAP.	ART.	% SAT.	V	A-V	O_2	CO	CI
16. W. B.	C	56	20	30	110	40	65	17.6	14.9	85	10.0	4.9	365	7.5	4.2
	C_o	50	16	28				17.6	16.8	96	11.8	5.0	365	7.3	4.1
	O(c)	85	22	58	130	50	75	17.6	14.0	80	8.2	5.8	544	9.4	5.3
	O_o	80	20	56				17.6	16.9	96	11.4	5.5	544	9.9	5.5
	OE	90	20	46				17.6	13.0	74					
17. C. E.	C	39	20	30	150	90	110	20.2	18.5	92	14.7	3.8	370	9.8	4.9
	C_o	35	15	26	150	90	110	20.2	20.2	100	13.2	7.0	370	5.3	2.7
	O(c)	38	18	28	150	90	110	20.2	18.1	90	16.0	2.1	370	18.0	9.2
	O_o	40	15	28	150	90	110	20.2	20.2	100	17.0	3.2	370	11.6	5.8
	O_oE	60	25	38	150	90	110	20.2	20.2	100	13.8	6.4	1060	16.6	8.3
18. B. L.	C	55	14	28	100	40	68	15.7	14.4	92	8.1	6.3	308	4.9	3.1
	C_o	53	18	30	90	35	62	15.7	15.2	97	8.8	6.4	308	4.8	3.0
	O(c)	85	27	43	90	40	63	15.7	9.6	61					
	O(p)	80	35	42				15.7	11.1	71	6.9	4.2	346	8.3	5.3
	O_o(p)	75	25	39	120	50		15.7	14.6	93	8.6	6.0	346	5.8	3.7
19. F. B.	C	25	8	12				17.3	16.0	93	12.0	4.0	281	7.0	4.0
	C_o							17.3	16.9	99					
	O(c)	34	8	15											
20. F. M.	C	28	10	17	100	55		12.5	10.8	86	6.2	4.6	240	5.2	3.8
	O_o(p)	35	18	25				12.5	11.8	94	7.5	4.3		5.6	4.0
	O_oE	60	25					12.5	11.9	95	5.5	6.4	530	8.3	5.9
21. G. W.	C	32	20	26	110	75	90	13.6	11.1	82	7.1	4.0	258	6.5	4.2
	C_o	26	12	20	120	75	95	13.6	12.7	93	8.7	4.0			
	O(c)	32	20	26	115	80	95	13.6	11.7	86	7.6	4.1		6.3	4.1
	O_o	30	18	24	130	80		13.6							
22. F. B.	C	35	12	20	100	60	75	13.0	11.1	85	6.0	5.1	313	6.1	3.7
	C_o	35	15	20	105	65	75	13.0	12.6	97	7.9	4.7	221	4.7	2.9
	O(c)	40	15	23	100	60	75	13.0	11.2	86	6.4	4.8	258	5.4	3.3
	O_o	35	10	20	110	60	80	13.0	12.8	98	8.1	4.7		5.5	3.4
	O_oE	40	10	24	125	80	80	13.0	12.7	98	6.6	6.1	442	7.3	4.5

23. E. W.	C	38	20	28	100	50	60	16.6	13.9	84	8.0	5.9	196	3.3	2.1
	C_o	34	18	27				16.6	14.8	89	9.0	5.8		3.4	2.2
	O(c)	45	18	30				16.6	14.5	88	8.9	5.6	250	4.5	2.9
	O_o	58	27	35				16.6	15.4	93	10.5	4.9		5.1	3.2
	O_oE	62	40	50				16.6							
24. O. B.	C	38	16	28	100	25	50	15.4	13.7	89	9.9	3.8	254	6.7	4.4
	O(c)	72	20	38				15.4	14.0	91	9.3	4.7	254	5.4	3.4
	O_o	70	18	35	130	30	65	15.4	14.8	96	12.2	2.6	254	9.8	6.2
25. E. O.	C	65	32	45				17.6	16.0	91	11.1	4.8	234	5.6	3.6
	C_o	63	30	44				17.6	16.5	94	9.1	7.4	234	3.2	2.1
	O(c)	65	30	46				17.6	15.9	90	10.0	5.9	378	6.4	4.2
	O_o	63	30	44				17.6	16.8	96	10.2	6.6	378	5.7	4.1
26. R. H.	C	60	30	40				16.4	14.1	86	7.9	6.2	226	3.6	2.1
	C_o	60	30	40				16.4	15.9	97	9.8	6.1	226	3.6	2.1
	CE	85	50	60				16.4							
	O(c)	70	35	50											
27. W. B. (a)	C	70	32	50	120	75	90	20.6	17.2	84	13.5	3.7	320	8.7	4.0
	C_o	62	32	47	115	70	85	20.6	19.0	93	15.6	3.4		9.4	4.2
	O(p)	60	29	42	110	65	85	20.6	18.1	88	13.6	4.4		7.3	3.3
	O_o	62	25	42			88	20.6	19.6	96	15.7	3.9		8.2	3.8
	O_oE	80	50	68									532		
W. B. (b)	C	42	25	32	100	50	75	20.1	17.1	85	13.7	3.4	345	10.1	4.7
	C_o							20.1	18.8	94	14.3	4.5		7.7	3.6
	O(p)	45	30	35				20.1	17.3	86	13.1	4.2	380	9.0	4.2
	O_o	42	24	30				20.1	19.0	95	14.1	4.9		7.9	3.6
28. D. E.	C	82	30	52	125	50	75	18.6	16.9	91	8.0	8.9	471	5.3	2.7
	C_o	80	25	51	125	60	78	18.6	18.3	99	10.8	7.5	471	6.3	3.2
	O(p)	82	40	55	120	60	75	18.6	17.0	92	9.4	7.6	525	7.0	3.6
	O_o	70	30	45	120	60	75	18.6	18.4	99	10.2	8.2	525	6.5	3.3

output was calculated utilizing the Fick principle. Control values were obtained before occlusion of the pulmonary artery by the inflatable cuff, and repeated following this maneuver. Control and occluded values were also determined with the patient breathing 100 per cent oxygen. In many patients, mild exercise was carried out and the effects noted. No attempt was made to attain absolute basal conditions during these previous studies, although a "steady study" was achieved.

The technique used in our laboratory has been presented.[2] In the series herein reported, all the patients had pulmonary disease with varying degrees of functional impairment, ranging from asymptomatic to completely incapacitating. In Table I are listed the subjects studied, with diagnosis and some of the standard pulmonary ventilatory studies. They are grouped according to diagnosis, as follows: (1) chronic diffuse, emphysema, with or without cor pulmonale; (2) bullous emphysema (complicated) or emphysema secondary to other pathologic processes; (3) granulomatous lung disease of various etiologies; (4) primary bronchiogenic carcinoma; and (5) combinations of cardiac disease involving the left ventricle and/or the mitral valve and pulmonary disease. Control catheterization findings were consistent with the grouping in that all of the patients with pulmonary disease without significant left ventricular or mitral valve disease had normal wedge (pulmonary capillary) or distal (lacunar) pressures. The latter pressure is that recorded beyond the inflated balloon. In all our studies, wedge and distal pressures have been almost identical. Patients with combined disease all had significantly elevated wedge or distal pressures. The subject of distal pressures and their significance will be discussed in a separate publication.

In most patients, complete unilateral occlusion was achieved as described. In some instances, however, as indicated in Table II, only partial occlusion of one lung was obtained, fortuitously or by intent. Usually, when the entire right or left lung was not occluded, this was due to the fact that the balloon was carried out beyond the desired point in the main subdivision of the pulmonary artery. In certain cases, because of severity of symptoms or initial marked elevation of pulmonary artery pressure, it was considered inadvisable to occlude the main branch.

RESULTS

In Table II are listed the pertinent findings in the cases studied. Unilateral occlusion was well tolerated in all but 3 patients (Cases 4, 16, and 18). Obvious difficulty had been anticipated in these 3. Case 4 was an individual with far-advanced emphysema who had become dyspneic even at rest. Case 16 had granulomatous lung disease of undetermined etiology (proved by lung biopsy), with dyspnea on slight exertion and marked impairment of ventilatory function. Case 18 was an elderly man with repeated episodes of chest pain, cyanosis, and shortness of breath. Chest x-ray studies suggested emphysema but pulmonary function studies were not consistent with this diagnosis. It was felt that he had developed the syndrome of alveolar-capillary block due to repeated pulmonary embolization.[9] In all 3, occlusion of the right main pulmonary

artery resulted in dyspnea, increased cardiac index, rise in pulmonary artery pressure, and fall in peripheral arterial oxygen saturation. The significance of this response is presented in the discussion.

On the other hand, in other patients (Cases 7 and 17) unilateral occlusion was tolerated much better than expected. Except in those instances noted, there were no subjective symptoms associated with occlusion.

Intracardiac Pressures.—In Fig. 1, the flow-pressure relationships are plotted for the *emphysema group* (chronic diffuse and bullous or secondary). In this group there is no correlation between the control main pulmonary artery pressure and the pressure following occlusion. The increase in flow through the unoccluded lung was attended by a significant increase in main pulmonary artery pressure in some patients (Cases 3, 4, 9, and 10b). However, in others (1, 2, 7a, and 7b), there was little or no increase. In 2 patients (Cases 10a, 12) the pressure actually fell during occlusion.

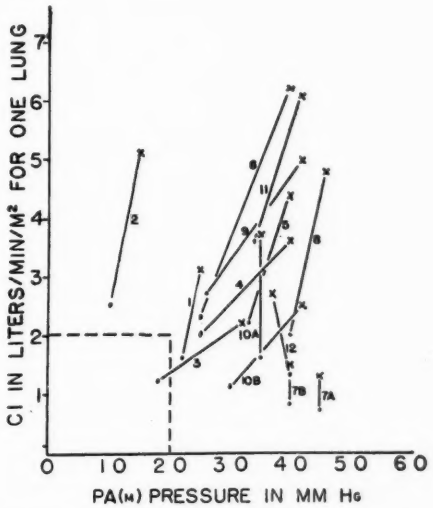

Fig. 1.—Flow-pressure relationships in patients with emphysema. Pulmonary artery mean pressure plotted against cardiac index for one lung. Symbols: ● = subject at rest; x = during unilateral pulmonary artery occlusion. Upper limits of normal at rest indicated by dotted lines.

In Fig. 2 are plotted the flow-pressure relationships existing in those cases of *granulomatous disease*. In every case, increase in flow by contralateral occlusion produced a marked increase in pulmonary artery mean pressure. Still further increases were produced with exercise. It should be pointed out that the increase in pressure was predominantly systolic with less change in the diastolic phase.

In Fig. 3 a similar plot is presented for those cases of combined *left ventricular heart disease and pulmonary disease*. In all but 1 case there was no pressure change with unilateral increase in flow. In the instance where pressure did rise significantly, granulomatous disease of the lung was also present.

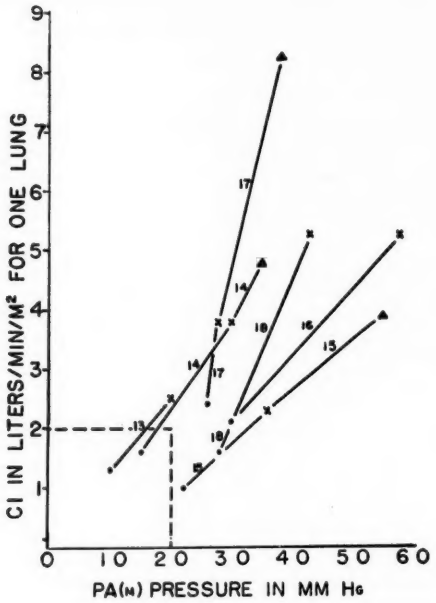

Fig. 2.—Flow-pressure relationships in patient with granulomatous disease. Symbols as before; ▲ = exercise during unilateral occlusion.

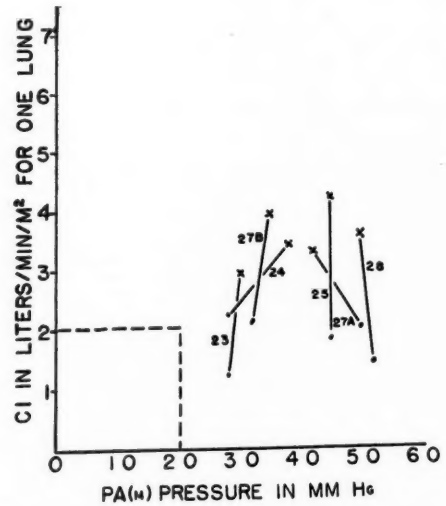

Fig. 3.—Flow-pressure relationships in patients with combined cardiac and pulmonary disease. Symbols as before.

The patients with carcinoma responded in a normal manner with a slight rise in main pulmonary artery pressure following occlusion. These pressures shown in Table II were within normal limits.

Patients 7, 10, and 27 were studied before and after definitive therapy. The first of these, before treatment for severe emphysema and cor pulmonale, had an end diastolic pressure in the right ventricle of 20 mm. Hg which decreased to 10 mm. Hg following therapy. Case 10 was studied before and after surgery for removal of bullae and Case 27 before and after mitral commissurotomy. It is notable that in the latter case, following commissurotomy, both wedge and pulmonary artery pressure had fallen and the response to occlusion was significantly altered.

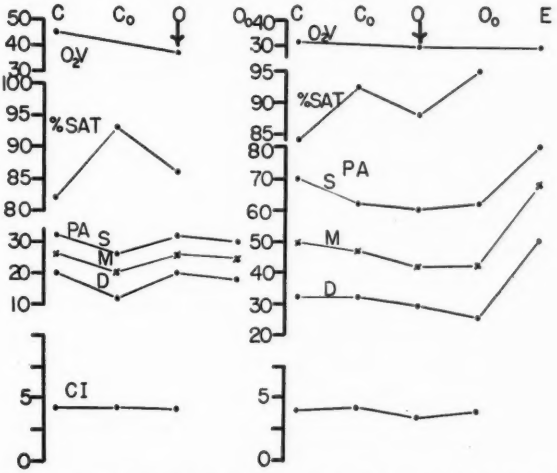

Fig. 4.—Effect of unilateral occlusion in 2 patients showing rise in arterial saturation during occlusion. Symbols: C = control; C_o = control on 100 per cent oxygen; O = unilateral occlusion; O_o = 100 per cent oxygen breathing during occlusion; E = exercise; O_2V = ratio of oxygen consumption to minute ventilation in cubic centimeter per liter; % Sat. = systemic arterial saturation; PA = pulmonary artery pressure in mm. Hg; S = systolic; M = mean; D = diastolic; CI = cardiac index in liters per minute per meter squared.

In Case 21 (*left*) the control values are normal for the O_2V and CI with slight elevation of the PA pressure and marked decrease in arterial O_2 saturation. On 100 per cent oxygen, the expected fall in PA pressure and increase in O_2 saturation occurred. With occlusion, however, the O_2 saturation rose as compared to the control value without other significant change. In Case 27 (*right*) initial values are normal for the O_2V but a markedly elevated PA pressure was present together with a somewhat increased CI and again a marked diminution in arterial O_2 saturation. On 100 per cent oxygen, there was a fall in PA pressure and increase in O_2 saturation and CI. With occlusion, the O_2 saturation rose significantly concomitant with a fall in PA pressure and CI toward normal as compared with control values. One hundred per cent oxygen caused a further increase in arterial O_2 saturation without other significant change and exercise was noted to cause a marked increase in PA pressure.

Peripheral Arterial Pressures.—No significant change was observed following occlusion.

Peripheral Arterial Oxygen Saturation.—In most cases, no change occurred. In at least 2 patients (21 and 27), peripheral arterial saturation increased following occlusion, as demonstrated by the hemodynamic response in Fig. 4. In both cases, a significant rise can be observed and, in Case 27, this was associated with a fall in cardiac index as well as pulmonary artery pressure. Both had disease involving predominantly the occluded side.

In Cases 4, 16, and 18, previously described, the opposite response occurred, namely, a fall in peripheral arterial oxygen saturation associated with a rise in cardiac index and pulmonary artery pressures. Fig. 5 demonstrates this response. All patients had severe chronic diffuse pulmonary disease and experienced dyspnea on occlusion.

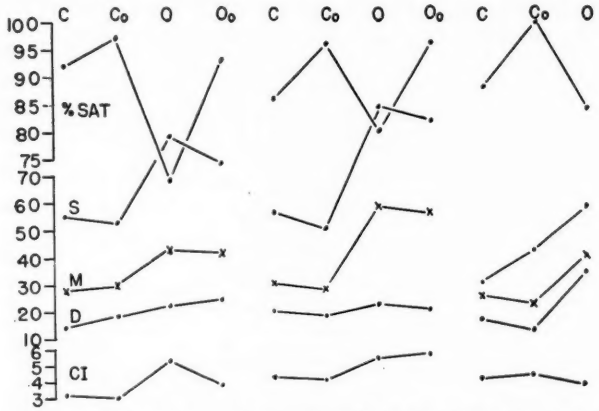

Fig. 5.—Effect of unilateral occlusion in 3 patients showing a decrease in arterial O_2 saturation. In Case 18 (*left*) the control O_2 saturation was slightly diminished, the PA pressure elevated, and the CI normal. On 100 per cent oxygen the arterial saturation rose to normal. With occlusion, the arterial O_2 saturation fell markedly associated with a sharp rise in PA pressure and CI. On 100 per cent oxygen, the saturation rose to control levels and the PA pressure and CI diminished.

Case 16 (*middle*) showed an initial decrease in O_2 saturation, elevated PA pressure and CI. On 100 per cent oxygen, the arterial saturation rose to normal levels. With occlusion the arterial O_2 saturation fell with a sharp increase in PA pressure and CI. One hundred per cent oxygen increased the saturation to normal once again but effected little change in pressure or CI.

In Case 4 (*right*), the depressed control O_2 saturation fell with occlusion again associated with a marked increase in PA pressure.

Cardiac Index.—This did not change significantly except in those cases described. Generally, for the purposes of this presentation, it can be assumed that there is approximately equal distribution of pulmonary blood flow to both lungs. However, in the presence of unilateral disease with atelectasis, a significant degree of shunting away from diseased lung may occur naturally.[10] In such cases, in which blood flow has already been redistributed, unilateral occlusion (of the diseased side) may only result in a relatively slight further redistribution.

Oxygen Consumption.—No significant change occurred except in those cases where dyspnea and resultant anxiety caused an increase.

Minute Volume.—Again there was no significant change although in many cases there was a slight rise. This is of particular importance since the occluded lung continues to ventilate but does not exchange gases.

O_2V.—Since the minute volume and over-all oxygen consumption did not change significantly and all gaseous diffusion was occurring in the unoccluded lung, the O_2V for that lung was approximately doubled.

Resistance.—As indicated in the figures showing the flow-pressure relationships, resistance was fixed in those patients with granulomatous lesions, presumably as a result of anatomic involvement of the small pulmonary vessels

by the disease process. In the patients with myocardial failure, however, despite an apparently increased resistance, this resistance was not due to pulmonary vascular change since increase in flow was not accompanied by increase in pressure. Thus, pulmonary resistance in these cases was greatly diminished following occlusion. In the emphysema group, many patients showed fixed resistance presumably on the basis of anatomic changes. In others, resistance was greatly diminished following occlusion. This suggests that structural changes in the pulmonary vessels need not have occurred despite initial high resistance.

DISCUSSION

The study of pulmonary hemodynamics in patients with chronic pulmonary disease yields widely varying results. The control pressure in the pulmonary artery may vary from normal to three times normal. It cannot be inferred, however, that significant pulmonary arteriolar disease exists solely on the basis of an elevated resting pressure. Nor can it be assumed that a normal or only slightly elevated pressure precludes the existence of disease. As has been demonstrated, sudden deletion of approximately 50 per cent of the pulmonary vascular bed, with resultant doubling of flow through the remaining portion, need not result in any significant rise in pressure whether the resting pressure is normal or even greatly elevated. Cournand[11-13] and Hickam and Cargill[14] have reported similar control elevation of pressures in patients with chronic pulmonary emphysema. Riley and co-workers,[15] by means of exercise, have demonstrated marked increase in pulmonary artery pressures in such cases. However, certain important differences exist between exercise as a test and unilateral occlusion. As pointed out earlier, the method described in effect increases flow (unilaterally) without over-all increase in cardiac output while, with exercise, over-all cardiac output is increased. Furthermore, factors involved in exercise such as increases in intrapleural pressures, changes in position, increased heart rate, reflex changes, metabolic changes, etc., differ considerably from those described.

Since pulmonary hypertension in emphysema is not necessarily due to arteriolar change per se, the cause of the hypertension must be re-evaluated. Increased intra-alveolar or intrapulmonic pressure in such patients presumably accounts to a considerable degree for the elevation of the pulmonary arterial pressure. Positive intrapleural pressures have been demonstrated to reach a level of 20 mm. Hg or more in emphysema.[16] These factors could increase resistance to pulmonary blood flow necessitating a rise in pressure to maintain circulation throughout all phases of the respiration and inherent vascular change need not be present. Perhaps the best explanation is that of the "critical closing pressure" discussed by Burton.[17]

As suggested by Lillienthal and Riley,[18] the concept of the "critical closing pressure" can be applied to pulmonary hemodynamics. According to this concept, in order to maintain blood flow through a vessel, the intraluminal pressure must be greater than those factors which would tend to collapse the vessel wall. Such factors include the elastic and muscular tension of the vessel wall itself as well as extravascular factors. It is presumed that in certain pulmonary disease states, such as emphysema, there may be a primary elevation of the

critical closing pressure because of the extravascular factors mentioned. Thus, the presence of pulmonary hypertension at rest does not necessarily imply anatomic pulmonary vascular change. Furthermore, a slight rise in pressure may open accessory channels allowing increases in flow out of proportion to the pressure increment.

The pressure response in patients with granulomatous disease was more predictable and confirms the findings of others.[4, 13, 19] In these cases, whether the resting pressure was normal or greatly elevated, occlusion, with unilaterally increased flow, produced a significant increase in pressure indicating considerable fibrosis of the pulmonary arterial tree, loss of elasticity, and hence a fixed resistance. The tremendous expansibility of the normal pulmonary vascular bed has been emphasized by Cournand[12] and in a previous publication.[2] Limitation of this distensibility by fibrotic pulmonary lesions can be fairly well defined by the method described.

It should be emphasized that the level of the resting pulmonary artery pressure does not necessarily reflect the status and distensibility of the pulmonary vessels. An elevated pressure is not necessarily a contraindication to resectional lung surgery. On the other hand, patients with normal pressures at rest may have no reserve with respect to accommodation to increased blood flow and are, therefore, poor risks for such surgery. Deletion of any segment of their pulmonary vascular bed would result in a significant increase in pressure.

In patients with combined pulmonary disease and cardiac disease involving the left ventricle or mitral valve, the evaluation of relative causes of disability may be extremely difficult. In the group studied, all had elevation of both pulmonary capillary (or wedge) pressure and pulmonary artery pressure. Only one, however, showed any significant rise with occlusion. Exercise in such cases increases the cardiac output. As a result, pulmonary artery pressure rises but this response does not aid in determining whether the increase in pressure is due to resistance in the left atrium or in the pulmonary vasculature. By means of occlusion, flow can be increased through a part of the lung without increase in over-all cardiac output thus facilitating the study of these two factors independently. It may be concluded from the cases shown that elevation in pressure was predominantly, if not entirely, due to increased left atrial pressure since no significant elevation in pulmonary artery pressure occurred with increase in flow. In these cases, the pulmonary vasculature is normal and can accommodate an increase in flow with a normal slight increase in pressure. In one case, however, the marked increase in pressure was evidence for the existence of increased resistance in the pulmonary vascular bed. Granulomatous pulmonary disease was subsequently proved by biopsy.

The importance of this relationship in patients with congenital and acquired heart disease, particularly in determining if secondary pulmonary arteriolar hypertrophy exists in patients with mitral stenosis, will be presented in a separate report.

Uneven Ventilation—Blood Flow Relationship.—The problems relating to ventilation-perfusion relationships in the lung have received much attention recently.[3, 5, 20, 21] It is now well appreciated that significant reduction in arterial

oxygen saturation may occur because of venous admixture in the lung. This situation exists when poorly ventilated alveoli are receiving a disproportionate perfusion. The result is failure of adequate oxygenation of blood from such areas and the equivalent of a right-to-left shunt. Methods for assessing the extent of such effects are indirect and often it is impossible to distinguish between faulty ventilation-perfusion relationships and disease of the alveolar capillary membrane with impaired diffusibility.

Utilizing the method described, it seems likely that localized areas of underventilation might be temporarily deprived of their pulmonary arterial supply. If the remaining unoccluded lung were normal, this maneuver should produce a rise in arterial oxygen saturation since all pulmonary capillary blood would be exposed to normal alveolar oxygen tensions. This procedure would have the added advantage of localizing such defective areas anatomically.

This effect is demonstrated in 2 cases with unilateral pulmonary disease and in 2 cases of diffuse pulmonary disease (Cases 3, 7, 21, 27). In these instances, occlusion of the pulmonary artery resulted in significant increases in arterial oxygen saturation. Paralleling this increase in saturation, there was observed a fall in pulmonary artery pressure and cardiac output. It seems apparent that areas of venous admixture were obliterated and over-all function improved.

In patients with bilateral severe chronic pulmonary disease, the opposite situation is also possible. Where arterial desaturation exists because of diminished ventilation-perfusion ratios, occlusion of one lung may accentuate the venous admixture occurring in the other. This situation obtains in 2 cases (Cases 8 and 9), where instead of a rise in arterial O_2 saturation there was a further fall. This response must be differentiated from that due to exceeding the maximal diffusing capacity of the unoccluded lung which will be discussed subsequently. In both instances, the further decrease in arterial O_2 saturation was associated with an increase in the pulmonary artery pressure, increased cardiac output and hyperventilation, necessitating deflation of the cuff in one case. On exposure to 100 per cent oxygen, both patients showed the expected rise to normal arterial O_2 saturation.

Diffusion.—Determinations of diffusing capacity of the lung at rest have shown wide variation and changes in diffusion have been demonstrated with exercise.[22] The maximal diffusing capacity of the lungs seems more promising as a basis for comparison in normal subjects and in those with pulmonary disease.[6, 23] Riley and co-workers have described such a method for maximal O_2 diffusing capacity, utilizing hypoxia and exercise. The factors limiting such determinations particularly in the presence of pulmonary disease may present considerable difficulty. Defects such as poor intrapulmonary mixing of gases, uneven ventilation-perfusion relationships, and circulatory failure may each cause a reduction in maximal diffusing capacity.

In this respect, of the cases studied, Case 7 is of particular interest. This patient initially presented a relatively normal resting arterial O_2 saturation and a normal cardiac index. Pulmonary artery pressure was elevated. Ventilation studies did not seem to indicate that obstructive pulmonary disease was the predominant difficulty. When occlusion of the right main pulmonary

artery was carried out, there was a marked fall in arterial O_2 saturation and an increase in pulmonary artery pressure and cardiac output. The O_2 consumption, however, did not rise despite marked dyspnea. On 100 per cent oxygen, arterial saturation rose to normal levels and cardiac output fell to normal. Although the factor of uneven ventilation blood flow has not been entirely excluded, it is felt in this instance that diversion of the extra blood flow through the unoccluded lung exceeded the maximal diffusibility of that lung due to specific disease of the alveolar capillary membrane. The increase in cardiac index and hyperventilation are explainable on the basis of hypoxia but, at the same time, indicate that neither cardiac output nor alveolar ventilation was a limiting factor in this patient's oxygen diffusibility. The normal resting arterial O_2 saturation rules out significant venous admixture. The maximal diffusing capacity in this patient therefore appears severely limited and can be exceeded merely by reducing the area of pulmonary membrane available for diffusion.

The application of this technique in determining maximum diffusing capacity of a single lung is obvious, particularly with respect to delineating the limiting factors in such determinations.

SUMMARY

1. Twenty-eight patients with chronic pulmonary disease have been studied by the method of temporary unilateral pulmonary artery occlusion using a special triple-lumen catheter with an inflatable cuff.

2. It is shown that the resting or control pulmonary artery pressure is not necessarily an index of the pulmonary circulatory reserve (expansibility). Without increasing cardiac output, approximate doubling of unilateral pulmonary blood flow may or may not be attended by significant increase in pressure, irrespective of the level of control pulmonary artery pressures.

3. It is suggested that in many cases of chronic pulmonary disease (without cardiac disease involving the left ventricle or mitral valve) pulmonary hypertension must be due to causes other than organic arteriolar disease, such as increased intrapleural and intrapulmonic pressures. The concept of a "critical closing pressure" is discussed in this regard.

4. In cases of mitral stenosis or left ventricular failure in combination with chronic pulmonary disease, this method has proved valuable in the study of pulmonary hemodynamics with particular reference to the status of the pulmonary arterioles.

5. The problem of uneven ventilation/blood flow relationships, where significant venous admixture exists, has been presented. It has been demonstrated that such areas can be obliterated by occlusion of the pulmonary artery to that segment. Resultant decrease in venous admixture has been demonstrated by a rise in arterial O_2 saturation.

6. Possible applications of temporary pulmonary artery occlusion in the determination of maximal O_2 diffusing capacity are discussed and particularly with reference to possible delineation of factors involved in such determinations.

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EXTRAPLEURAL THORACOPLASTY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

An Eight- to Twenty-One-Year Follow-up Study

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UNTIL the discovery of streptomycin, bed rest and collapse therapy were the mainstays in the treatment of pulmonary tuberculosis. Of the various forms of collapse therapy, artificial pneumothorax and extrapleural thoracoplasty were the procedures upon which we depended for a number of decades. During that period many thousands of extrapleural thoracoplasties of diverse types were performed. As the adjuvants to surgery developed and surgical technique improved, it was possible to obtain increased pulmonary collapse, and there was a decrease in surgical complications; consequently, operative results were decidedly better.

With the introduction of the various antituberculosis drugs, a sudden change in surgical therapy developed. Collapse therapy quickly yielded to drug therapy. When a cure was not obtained by the use of drugs alone, then pulmonary resection became the procedure of choice. It was the abruptness of this change which, in a great measure, robbed collapse procedures, especially thoracoplasty, of the opportunity to demonstrate the good results they might achieve when used in conjunction with the newly found drugs. So few collapse procedures are being performed in this country today, that it will be many years before any one center will accumulate sufficient data to compare the results of the various collapse methods with those obtained by excisional therapy, when both have been performed with the aid of the new therapeutic agents.

Because of these circumstances, we feel that it is of the utmost importance to evaluate precisely the long-term results of collapse methods and especially extrapleural thoracoplasty. The known chronicity of pulmonary tuberculosis and its proclivity for reactivation many years after apparent cure make only long follow-up periods of value. With this knowledge in hand, we should be able to postulate to some degree what our long-term results would be were thoracoplasty performed with the additional benefits that could be derived from the simultaneous use of our antituberculosis drugs. Only in this way will we be able to compare our long-term results of excisional therapy (when we have them) with what could be expected from collapse therapy if performed under a similar drug therapy regimen.

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A number of reports in the foreign literature give evidence that collapse methods, such as pneumothorax and thoracoplasty, when aided by an antituberculosis drug regimen give immediate and short-term results comparable to the excellent results achieved by excisional surgery.

The early and late results of 90 thoracoplasties performed on the ward service at Montefiore Hospital, in New York City, from 1935 to 1940, have already been reported.[1, 2] To these have been added the data of an additional consecutive 90 patients from the same service operated upon by the authors from 1940 to 1947. (It was entirely accidental that there were an equal number of patients in each series.) The total number of patients treated by extrapleural thoracoplasty, primarily for pulmonary tuberculosis, is 180. Except for a few cases in the first series, those patients in whom an empyema overshadowed the pulmonary disease were excluded from this report.

The extent of the follow-up was such that it took a number of years to complete. We have a 100 per cent follow-up, i.e., the status of every patient has been ascertained. Four patients who had been operated upon before 1941 were last contacted in 1953. The status of the remaining number was determined in 1954, 1955, or 1956, the date of preparation of this paper. Most of the follow-up data were obtained from the Altro Work Shops, Inc., from the Montefiore Hospital Follow-Up Clinic, from Board of Health Clinics, and various other clinics in New York City, as well as those in other cities and states, and from private physicians. In only a small number of cases were the reports obtained directly from the patients. These were patients operated upon many years ago, who were no longer under routine medical care. The reports on sputum examinations were based on cultures; a few were made on concentrates.

We have been more concerned with those patients in whom thoracoplasty was unsuccessful rather than with those who were cured. We have analyzed the case histories with a view to determining the reason for failure to cure and how this cause for failure could be avoided today. When these patients were operated upon, there was no alternative good procedure which could be used. Penicillin became available to only a few patients in this series; none had the advantage of the antituberculosis drugs; and many of the aids to surgery and to anesthesia which are used routinely today were not yet available.

There were 9 postoperative deaths; 6 of these occurred in the first series of 90 patients and 3 in the last 90. Of the first 6 deaths, 3 were due directly, or indirectly, to a pleural tear occurring during operation. These tears were packed with gauze, and the complications resulted eventually in a fatality. The fourth death was caused by a phlegmon of the back. The fifth patient died thirty minutes after operation and, at autopsy, a contralateral atelectasis was found. The sixth death was that of a patient who died twelve hours postoperatively with convulsions, tachycardia, hypertension, and delirium. At autopsy, an unsuspected pheochromocytoma of the adrenal was found. Of the 3 deaths in the second group of 90 cases, one was due to operative shock and occurred twelve hours after a first-stage procedure; in the other 2, the causes of death were unexplained. One, a 46-year-old man, was found dead on the third postoperative day after a two-day uneventful course; the

other, a 40-year-old woman, died suddenly on the first postoperative day. Post-mortem examinations were performed on both these patients, but in neither one could a cause of death be found. It is fair to assume that, today, with the use of the antibiotics and a more rational handling of pleural tears, at least 4 of the first 6 deaths might have been avoided.

There were 35 additional deaths in the entire series. In 10 of these patients, the thoracoplasty had been successful in arresting the pulmonary disease; death was caused by carcinoma of the stomach in 3 patients; suicide in 2; a brain tumor in one; a carcinoma of the breast in another; hemorrhage from bronchiectasis in one; a coronary occlusion in another; and the tenth patient died from hypertensive heart disease complicated by a cerebral vascular accident.

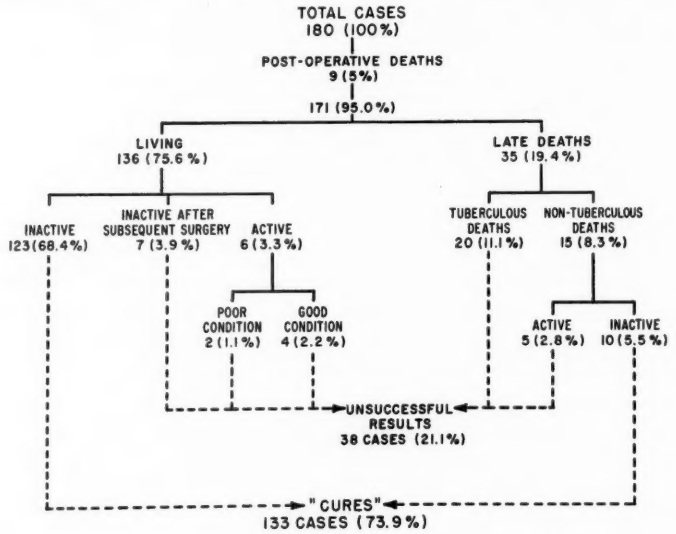

Fig. 1.

Post-mortem examinations in several of these patients substantiated the diagnosis of a clinical cure of the pulmonary tuberculosis. These 10 patients had been classified as arrested cases for from two to fourteen years, an average of 7.6 years. Death occurred from two to sixteen years after operation, with an average of 9.8 years. In the remaining 25 cases, death was caused by progressive pulmonary tuberculosis in 20 patients. Although the other 5 patients had active tuberculosis at the time of death, the fatal issue was attributed to suicide in 2; to a gastric carcinoma in one; to a coronary occlusion in another; and in the fifth case of this group, it was attributed to cardiorespiratory insufficiency which followed a contralateral thoracoplasty performed twelve years after the original procedure.

Of the entire series of 180 patients, 136 are still alive. One hundred and thirty of these patients are considered cured. Seven patients in this group required additional surgery to effect the cure and therefore we have classified

them as poor results of thoracoplasty. Another 6 patients still have active tuberculosis; of these, 4 are in generally good condition and 2 are in poor condition. Of these 4 patients with a positive sputum, one had a spread to the other lung after the first-stage thoracoplasty and has remained in good general condition over a period of thirteen years; the second was operated upon for "cavitary and disseminated disease" which had progressed rapidly during the preoperative period of bed rest. Nine years after operation, she had a panhysterectomy, and at present feels well but has a positive sputum on culture. The third patient was considered as a case of advanced disease because of a 5 cm. cavity, plus disseminated disease and contralateral emphysema. In spite of positive sputum cultures, he works as a musician on a full-time basis. The fourth patient, in good general condition but with a positive sputum, was also considered a poor subject for thoracoplasty because of multiple cavities, contralateral disease, and pleural effusions. He also had a Kaposi sarcoma of the foot. Eight years after operation, which was performed as "his only chance," he is attending college.

Of the 2 patients in poor condition, one refused sanatorium care postoperatively and returned to work, driving a taxi, soon after operation. Four years later he had a reactivation, with generalized spread of his disease and, when last contacted, he was a patient in a sanatorium. The second patient, a woman, also refused postoperative sanatorium care. She developed renal disease, requiring a nephrectomy. She is semi-invalided at the present time.

Excluding the 9 postoperative deaths, we have 38 patients in whom thoracoplasty was unsuccessful. This includes the 20 patients who died from progressive pulmonary tuberculosis, the 5 patients with active tuberculosis who died from other causes, the 7 patients who required further surgery to effect a cure, and the 6 patients who today have active disease. We have analyzed these cases in an attempt to determine why thoracoplasty was a failure, and we have classified the possible causes thereof. A number of the patients showed more than one possible cause for failure of their thoracoplasty, therefore, the total number listed in Table I is greater than the number of unsuccessful cases.

TABLE I. POSSIBLE CAUSES FOR THORACOPLASTY FAILURE

Contralateral disease	20
Giant cavities	11
Multiple, middle, or lower lobe cavities	9
Delayed stages of thoracoplasty	7
Patients in very poor condition	6
Incomplete surgery	6
Endobronchial disease	4
Postoperative spreads	4
Bronchiectasis and fibrosis	2
One-stage procedure	2
Refused postoperative sanatorium care	2
Oleothorax and empyema	1
Progressive nonpulmonary tuberculosis	1
No apparent cause	3
Total	78

In our previous paper, we stressed the fact that many late reactivations after thoracoplasty occur in the upper lobe of the opposite lung. They appear

to be independent of the disease which, originally present on the operated side, has remained arrested. Subsequent careful study of the preoperative roentgenograms frequently reveals that contralateral disease was present at the time of thoracoplasty (twenty of the unsuccessful thoracoplasties). Theoretically, such a reactivation should not mean that the operation was not a success, but we have classified these patients as having had unsuccessful thoracoplasties, in which group they are listed.

In our series, operation was not performed unless the contralateral disease was presumed to be stable and inactive. Most of these patients did not have the benefit of tomographic examination, therefore, the status of the contralateral pulmonary disease could not be established as accurately as it is today. Now, with the use of the antituberculosis drugs, contralateral disease can be better controlled prior to, and after, surgery and the likelihood of late reactivation will be lessened.

It was common knowledge that giant cavities and tension cavities were not readily closed by thoracoplasty. Yet, having nothing better to offer (we did not find the Monaldi suction drainage procedure of much value), many of them were treated by thoracoplasty. In a very small number was closure obtained. These cavities, both giant and tension, usually secondary to endobronchial tuberculosis, have now become a rarity with the use of the antituberculosis drugs.

Thoracoplasty was unsuccessful in 9 patients with multiple cavities or cavities in the middle or lower lobes. A small number with this type of pathologic condition were cured by operation, but the percentage of successful cases was so small that thoracoplasty would not have been considered as a method of treatment had some other procedure been available.

When infection of the wound occurred in the days before penicillin was available, the wound had to be opened widely and packed in order to control the infection; the second-stage operation could not be performed until the wound was well healed. This frequently took from six to eight weeks. Because of this delay, very little additional collapse was obtained after the second stage and cavities, therefore, ofttimes remained open. In our series, there were 7 patients in whom the second stage of the thoracoplasty had to be unduly delayed because of wound infection. Today this operative complication rarely, if ever, occurs, and a two-stage thoracoplasty can usually be completed in ten days. Furthermore, the various types of thoracoplasty which have been evolved in the past few years, using various methods of plombage in conjunction with rib resection or even leaving the ribs in situ, can be performed in one stage.

Six patients in our series were operated upon as a "last resort." They were in poor general condition; they had extensive disease; and despite all measures available at the time, they could not be "built up" sufficiently to be regarded as good operative risks. Two of these patients are alive today, but they do have positive sputum. Both had had a destroyed lung, for which thoracoplasty, at that time, was the only procedure that could be used. They both live quite active lives, and we can assume that were it not for the benefits derived from the collapse obtained by thoracoplasty, they would have died before

this. They are, however, considered as poor results of thoracoplasty because of positive sputum. It is most unusual today to have to operate upon a patient in poor condition. Practically all of the patients who have pulmonary tuberculosis respond, at least to some degree, to the antituberculosis drugs; the tempo of the disease is thus lessened and the patient can be the better prepared for surgery.

There were also 6 patients in whom the poor result of thoracoplasty could be ascribed to incomplete surgery. In 4 of these patients, a deterioration in the general condition prevented the second-stage operation; one patient committed suicide; and the sixth patient refused further surgery, became psychotic, and was discharged for disciplinary reasons.

Today, with the use of the antituberculosis drugs, endobronchial disease and its complications are well controlled and are not a problem in surgery. Not all the patients in our series were bronchoscoped, therefore, many cases of endobronchial disease were not diagnosed. Four patients, known to have endobronchial disease, had unsuccessful thoracoplasties.

The spread of pulmonary tuberculosis to other segments of the same lung or to the other lung was one of the hazards of thoracoplasty. Such a spread usually delayed the second-stage procedure for so long a period that it rarely was effectual. In some instances, the postoperative spread was the beginning of the ultimate fatal progressive tuberculosis. Today, under drug therapy and with improved surgical technique and intratracheal anesthesia, postoperative spreads are rare indeed.

Each of the remaining classifications listed under the reasons for the unsuccessful thoracoplasties embraces too few cases to justify discussion. It is apparent, however, that today these patients would not be treated by collapse therapy, and the use of the antituberculosis drugs would in all probability eradicate the reason for failure.

In only 3 of the 38 cases of unsuccessful thoracoplasty were we unable to find some apparent reason for the failure of operation. With this past experience to guide us and with the use of the antituberculosis drugs and our improved techniques in surgery and anesthesia, if thoracoplasty were performed today upon a well-chosen series of patients, a very high percentage of permanent cures would be achieved.

The following objections to thoracoplasty have been raised: (1) the operation causes a deformity; (2) some patients have persistent cough and expectoration due to the fibrosis and bronchiectasis which are the end results of pre- and postoperative healing; (3) the diseased pulmonary tissue is still present in the body and may not undergo complete healing, thereby becoming a source for later infection; and (4) there is a reduction in pulmonary reserve and the patient later succumbs to cor pulmonale.

In our follow-up study, the question of deformity and arm and shoulder motion was thoroughly investigated. Scoliosis, as seen on the roentgenogram, may appear to be much more marked than it is clinically. Many of our patients had a mild degree of scoliosis, but in none was it incapacitating and, as far as we could determine, it was never a cause of mental disturbance. In both

sexes the ordinary clothing gave adequate concealment. Arm and shoulder mobility seldom was affected seriously. The occasional severe scoliosis occurred in the patients who developed wound infection. Today, as previously stated, this complication rarely ensues.

The study of those patients in whom cough and expectoration (with negative sputum) persisted revealed that considerable fibrosis and bronchiectasis was probably present before operation. This was visible on their preoperative roentgenograms. Today the antituberculosis drugs prepare the patient for operation so much more quickly that fibrosis and bronchiectasis are minimal.

From the few post-mortem examinations available to us on patients who died of other causes after cure of their tuberculosis, we can state that thoracoplasty did result in a pathologic, as well as a clinical, cure.

There has been no severe reduction in pulmonary reserve in any patient who had had uncomplicated unilateral pulmonary disease; and some of these patients are alive up to twenty years after thoracoplasty. One patient who had been well for seventeen years has developed cor pulmonale, but she had bilateral collapse therapy; thoracoplasty on the right, and oleothorax on the left. Cor pulmonale did develop and was the decisive cause of death in a few patients in whom the thoracoplasty was unsuccessful in preventing the progression and spread of the disease; but in those patients in whom a unilateral tuberculosis had been cured, there were no late deaths from cor pulmonale.

It is interesting to note that there were 4 patients in our series in whom the conversion of sputum did not occur before the eighth, ninth, tenth, and thirteenth postoperative years, respectively. When this conversion took place, it did so without the addition of any other form of surgical therapy and without the administration of any antituberculosis drugs. Curiously enough, once the sputum became negative in these late conversions, it remained so.

From our study, we can report that thoracoplasty for pulmonary tuberculosis performed in the pre-antibiotic era resulted in 73.9 per cent cures. These results are, in the main, similar to other reports which have recently appeared in the literature. We believe we are justified in postulating that a much higher percentage of good results would be obtained were a similar group of patients operated upon today with the aid of all the new drugs and techniques. And were we to eliminate from our present-day operations those patients with pathologic conditions which we now know are not amenable to collapse therapy, we believe our results would approximate those of resectional therapy.

It is significant that the ideal pathologic condition for thoracoplasty is also an ideal case for resectional therapy; and in our enthusiasm for the latter type of operation, thoracoplasty is rarely considered as a method of treatment. But it is also significant that as the follow-up period lengthens upon patients who have had resectional therapy, an increase in the number of reactivations has been observed. When thoracoplasty is performed only for suitable types of disease, it will yield a high cure rate; therefore, it should not be discarded from the surgical treatment of pulmonary tuberculosis. The other methods of surgical therapy will have to equal or better the percentage of cures after a prolonged follow-up period in order to justify their use in preference to thoracoplasty.

DISCUSSION

One of the ordinary methods of reporting the results of surgical treatment is shown in Fig. 1. This gives the status of all patients on a specific date, the last follow-up date. This does not give the complete picture for (1) the results in cases with varying lengths of follow-up are considered with equal significance, and (2) temporary exacerbations with positive sputum are not discernible if the patient reconverted without additional surgery.

In analyzing the results of a chronic disease such as pulmonary tuberculosis, with its many periods of exacerbation and remission, we have tried to portray in Fig. 2 a year-by-year picture of the results. This method is also not perfect because it can only be used for those patients who remain alive. Those who died, either from tuberculosis or from a nontuberculous disease after being considered as cured, cannot be carried in the figures beyond the year of death.

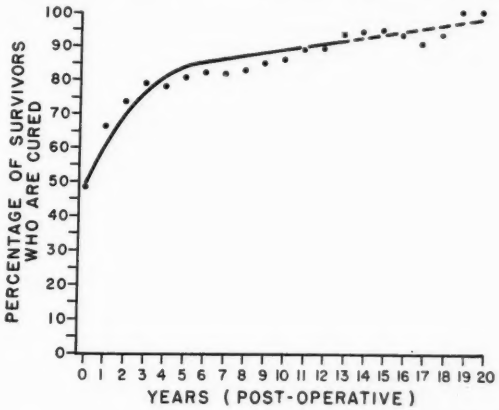

Fig. 2.

This then shows the status of only the living patients in each postoperative year. If a patient had only one positive sputum in a year, he was considered as "positive" in that year. A number of patients, although clinically well, have had an occasional report of a positive sputum over the years. Fig. 2 also gives expression to such incidents as the late conversion which occurred in 4 patients. In Fig. 1 they are considered as cured, but in Fig. 2 they are not classified as such until the year of persistent negative sputum.

Although neither of these charts gives the complete picture of the results, we believe that they supplement each other, thereby presenting a more accurate expression of what thoracoplasty accomplished in the treatment of pulmonary tuberculosis in the pre-antibiotic era.

SUMMARY

1. A complete analysis of thoracoplasties performed upon 180 patients at the Montefiore Hospital, New York City, from 1935 through 1948 is presented.

2. There has been a 100 per cent follow-up.

3. The cases in which an unsatisfactory result was obtained have been analyzed in an attempt to determine the cause for the poor outcome.

4. Seventy-three and nine tenths per cent of the patients were cured by thoracoplasty alone. Surgical mortality was 5 per cent.

5. Some of the commonly held objections to thoracoplasty are discussed. They were not found to be prevalent in our cured group of patients.

6. The results which might be obtained by thoracoplasty, if performed today, are postulated.

7. Thoracoplasty performed upon a well-chosen group of patients should yield long-term follow-up results comparable to the results thus far obtained from resectional therapy, for which similar lengthy follow-up periods are not as yet available.

8. There are indications for the use of thoracoplasty in the treatment of pulmonary tuberculosis and this procedure should not be discarded.

9. The results are charted by two methods: (1) the end result on the last follow-up date, and (2) the status of the survivors in each postoperative year.

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THE SURGICAL TREATMENT OF PULMONARY HEMORRHAGE IN TUBERCULOSIS

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PULMONARY hemorrhage is a serious complication of tuberculosis because it frequently causes spread of the disease. Hemorrhage which persists or recurs or is large in volume may be a threat to life itself. This paper concerns itself, in part, with the reporting of resectional surgery performed without complications in 3 cases in which the risk of operation was deemed desperate.

CASE REPORTS

The following 3 case histories summarize our recent surgical experiences with this problem. In 1952, one emergency pneumonectomy was performed. In 1954, one pneumonectomy and one right upper lobectomy were performed.

CASE 1.—This 26-year-old Negro man's first indication of pulmonary tuberculosis was a single hemoptysis of 50 c.c. of blood in January, 1951. Examination revealed a large left upper lobe cavity, and bilateral pneumonic infiltrations. The stained smear of the sputum was positive for tubercle bacilli. The patient received PAS and streptomycin daily for 18 months. During this period a left phrenic crush and pneumoperitoneum were performed, and the patient was kept at bed rest. He then left the hospital against advice.

Two months later, after 3 days of recurrent hemoptyses, he was re-admitted to the hospital. Examination disclosed findings similar to those found at the time of the first admission. Bleeding continued. On the sixteenth day after admission, following recurrent hemoptyses amounting to 3,300 c.c. in 5 days, and further involvement of the right lung, left pneumonectomy was performed. Chemotherapy consisted of isoniazid and PAS. The right lung infiltrations cleared slowly. The patient was discharged 9 months postoperatively, and chemotherapy was continued for a total of 22 months. Thereafter, the patient worked as a carpet layer.

Roentgenograms of March, 1954, and March, 1955, were identical in appearance and showed no evidence of tuberculous activity. Sputum cultures have been negative for more than 2 years.

This patient's x-ray studies *do* show a thin-walled "cyst" 2 cm. in diameter in the right middle lobe (Fig. 1). Its size changes with respiration. Attempts to demonstrate a focus of tuberculosis have been unsuccessful, even from cultures taken during and after hospitalization for pneumococcal pneumonia of the right middle lobe, March to May of 1955.

Pathologic examination of the resected left lung revealed two aneurysms of branches of the pulmonary artery in the walls of cavities. Bacteriologic studies showed complete sensitivity to streptomycin (no growth in medium containing 3.5 mcg. streptomycin per millimeter), despite 18 months of streptomycin and PAS given previously.

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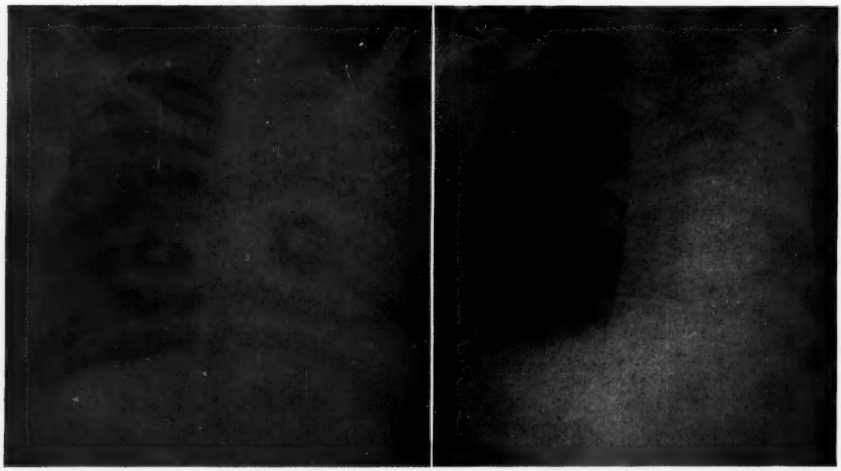
CASE 2.—In December, 1952, a 22-year-old Negro man was admitted to Bellevue Hospital Chest Service for treatment of pulmonary tuberculosis. Examination showed a left upper lobe cavity measuring 6 cm. in diameter, and diffuse involvement of the left lung and of the upper lobe of the right lung. Streptomycin and PAS were given daily for 3 months when the patient left the hospital against advice. He was re-admitted with a single small hemoptysis one month later, but stayed only one day. Nine weeks later he was re-admitted without hemoptysis. He was transferred to a sanatorium for treatment, and received streptomycin, isoniazid, and PAS. He was discharged 11 months later, April 1954, without further medication.

He was admitted to the hospital again on June 10, 1954, with a history of an hemoptysis of 200 c.c. This had been preceded by 2 weeks of cough and fever. Large cavities were present in each lobe of the left lung. In the next 14 days, bleeding totalled 1,300 c.c. There was spread to the right lung. Left pneumonectomy was performed on June 24, 1954, 17 months after the first admission for tuberculosis. The pneumonia in the right lung cleared rapidly. He was treated with streptomycin, isoniazid, and PAS. Sputum studies in August, 1954, were negative. The patient left the hospital against advice in September, 1954.



A. *B.*

Fig. 1.—Case 1. *A*, Preoperative roentgenogram in August, 1952, illustrates the extent of bilateral tuberculosis.
B, Postoperative roentgenogram in May, 1955, shows right middle lobe "cyst."

This patient has continued to have sputum negative for acid-fast bacilli on smear and culture. When last seen in May, 1956, he was well, and the x-ray examination of his chest showed no new or active disease.

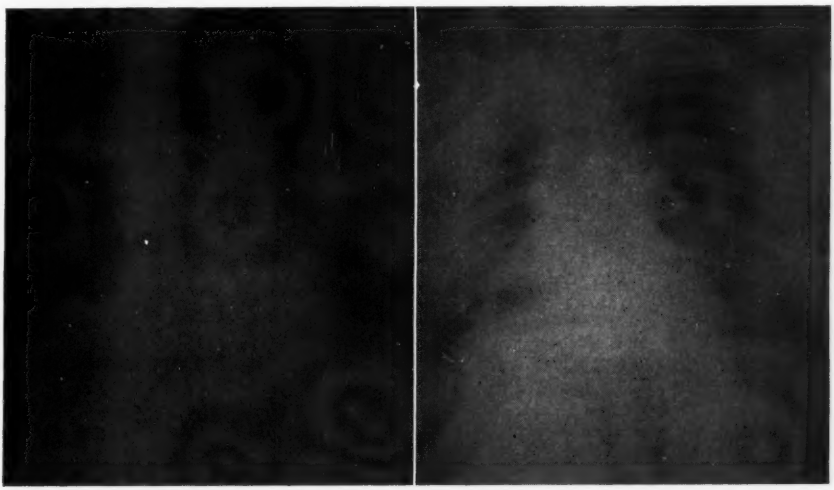
The removed lung revealed large cavities in each lobe and erosion through the wall of a branch of the pulmonary artery to the left upper lobe. There is no record of sensitivity studies on the tubercle bacilli from this patient.

CASE 3.—A 47-year-old Negro man entered the Bellevue Hospital Chest Service in August, 1954, following 3 weeks of hemoptysis. In the first 2 weeks, the sputum was blood streaked; in the last week there was hemoptysis of 300 c.c. a day. He was known to have had tuberculosis for one year. He had been treated in another hospital with streptomycin, isoniazid, and PAS daily for 5 months, and with an additional 3 months of isoniazid alone before leaving that hospital against advice. A 2-month interval without therapy and at full activity, including full-time employment as a store clerk and messenger, preceded this admission to Bellevue Hospital.

Examination revealed a large right upper lobe cavity, caseous pneumonic processes in the right middle and lower lobes, and early infiltrates in the left lung (Fig. 2). Recurrent hemoptyses and increased spread to the same and to the opposite lung led to right upper lobectomy with concomitant four-rib thoracoplasty[1] one month after admission, on Sept. 28, 1954. There were no pulmonary or pleural complications postoperatively. Chemotherapy postoperatively was viomycin and PAS, discontinued because of an elevated BUN, followed by pyrazinamide and PAS. The sputum became negative in November, 1954, 2 months after surgery.

This patient was examined in November, 1955. Five sputum cultures were negative for acid-fast bacilli. A roentgenogram of the chest showed no evidence of active disease.

The removed right upper lobe contained a large cavity filled with blood clots. A specific bleeding site was not demonstrated. In drug-susceptibility tests, the tubercle bacilli from the sputum cultures on admission showed 4+ growth in media containing 15 mcg. of streptomycin per milliliter. There was 2+ growth in media with 200 mcg. of streptomycin per milliliter.[4]



A. *B.*

Fig. 2.—Case 3. *A*, Preoperative bedside roentgenogram showing extensive tuberculosis, bilaterally.

B, Roentgenogram made 7 months after right upper lobectomy and partial thoracoplasty of ribs 2, 3, 4, and 5.

COMMENT

The sequence in each case was inadequate and interrupted chemotherapy, when the patients left the hospital against advice. On re-admission, each patient was found to have a large, thick-walled cavity. There was positive sputum in large quantity, and there was reason to suspect resistance to previous forms of chemotherapy. These patients appeared to have established the pattern of recurrent or persistent bleeding in large quantities, and had deteriorated to the point where medical therapy alone was judged to be inadequate to arrest bleeding and to preserve life.

Review of Fatal Cases of Pulmonary Hemorrhage.—The records and roentgenograms of 21 patients who died as a direct result of pulmonary hemorrhage in the 10-year period, 1945 to 1954, on the Bellevue Hospital Chest

Service were reviewed (Fig. 3). One exsanguinated, 10 died of asphyxia, and 10 died of toxicity and hypercapnea following spread of tuberculosis (Fig. 4).

The facts indicated that age and sex were of no significance. The known duration of tuberculosis in these patients had no bearing on the quantity or frequency or outcome of the hemorrhage. The history of previous hemorrhage had no relation to the severity of the current hemorrhage.

The local characteristics of the lesions appeared to be more important. All patients had cavities, which were of various sizes, and located in upper or lower lobes, or both. All these patients had cavities with thick walls.

Thus, it would appear that the setting for continuing hemorrhage is to be found in the patient who has a thick-walled cavity.

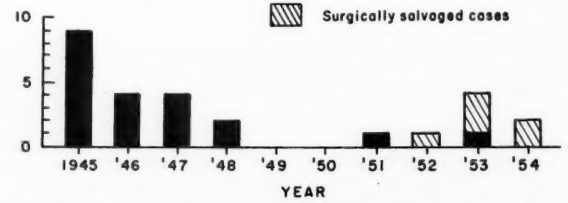


Fig. 3.—The incidence of deaths from pulmonary hemorrhage on the Belevue Hospital Chest Service, 1945 to 1954, with indication of patients saved by surgery.

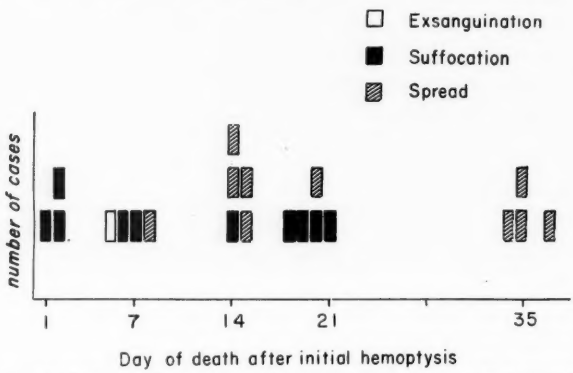


Fig. 4.—The causes and time of death from pulmonary hemorrhage in 21 patients, 1945 to 1954.

DISCUSSION

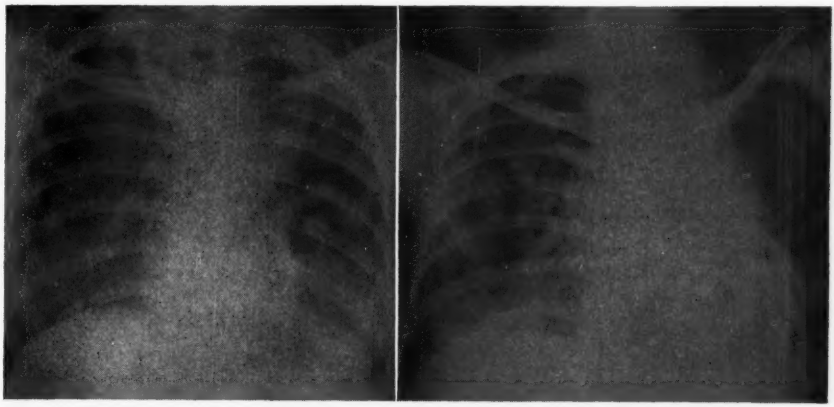
Therapy of the Patient With Hemoptysis.—We have no statistics of the degree and severity of hemorrhage in patients who were treated successfully by therapy other than surgery. Certainly the majority of pulmonary hemorrhages cease spontaneously or respond to nonsurgical therapy after a short period of time. This is to be explained convincingly to patients with pulmonary hemorrhage upon admission.

The patient is positioned with the bleeding side dependent, and the head of the bed lowered, to aid cleansing the tracheobronchial tree while limiting gravitational spread. Coughing is permitted and encouraged at intervals. Excessive coughing is to be avoided mainly by patient cooperation, but with

the use of barbiturates and 15 or 30 mg. doses of codeine every few hours if necessary. Heavy sedation or narcosis is avoided. Oxygen by tent or nasal catheter is used if necessary. Blood is typed and cross-matched. The patient's response to therapy is observed and noted.

Pneumothorax has been attempted, with the hope of arresting bleeding by changing the dynamics in the cavity. It was not successful when tried in several of the 21 cases cited, because of pleural symphysis. This experience presumably explains the infrequent attempts to employ pneumothorax in this group.

Two patients were treated for hemorrhage with thoracoplasty. In one of these the hemorrhage stopped after the thoracoplasty had been performed. Pneumonectomy was performed 2 years later for a destroyed lung. In the other, the hemorrhage finally was controlled after thoracoplasty and revision. This patient died one year later as a consequence of diffuse bilateral spread of tuberculosis and subsequent fatal hemorrhage (Fig. 5).



A. *B.*

Fig. 5.—*A*, Admission roentgenogram of 35-year-old patient, whose subsequent pulmonary hemorrhage was controlled by (*B*) two-stage thoracoplasty. Death occurred one year later from hemorrhage from cavity in superior segment of right lower lobe.

Indications for Resectional Surgery.—It is of interest to attempt to define the indications for resectional surgery in pulmonary tuberculosis complicated by hemorrhage. From the experience presented, the indication for resectional surgery is limited to the instance of the patient with the thick-walled cavity who continues to have pulmonary hemorrhage in large amounts, and in whom a fatal outcome seems certain without operation.

The decision to operate and the timing of surgery are difficult, and may be determined only after repeated observations of the patient as hemorrhage continues. In order to consider excisional surgery, the cavitary disease must be unilateral. The contralateral disease must be recent.

In the 3 patients upon whom a decision to operate was made, it was known that there was unilateral cavitary disease. The episode of bleeding consisted of three or more large hemoptyses. There was spread to the other side. There

was increasing respiratory distress. The patients appeared weaker and more ill in every way. In the desperate situations in which these judgments for resectional surgery have been made, the question of whether the contralateral spread was tuberculosis or blood pneumonia was not of sufficient importance to deter surgical intervention.

Individual case reports of pneumonectomy for the control of pulmonary hemorrhage in tuberculosis, by Ryan and Lineberry[3] and Ross,[2] applied similar criteria of selection and timing for excisional surgery.

Contraindications: We do not advocate excisional surgery in pulmonary hemorrhage except as a lifesaving measure to protect a patient from asphyxia and death. Although the coincident removal of the major focus of disease in each instance was a major dividend, we believe that properly timed elective resections will yield the best results.

It is conceded that advanced bilateral caseocavitary tuberculosis, pulmonary insufficiency, and other usual contraindications will obviate attempts at surgical therapy in many instances.

The Operation.—At each operation an experienced anesthetist gave a rapid, deliberate induction and employed a cuffed Magill endotracheal tube. The usual lateral decubitus position of the patient was employed with the head of the table lowered. It was felt that the theoretical advantages of a bronchial block or a divided, selectively occluding, endotracheal tube, or of an unusual position of the patient on the operating table, were unnecessary. Surgical technique was directed toward rapid occlusion of the bronchus to the involved lobe or lung.

SUMMARY

Most instances of pulmonary hemorrhage secondary to tuberculosis cease with proper medical therapy.

The setting for recurrent and uncontrollable hemorrhage is to be found in the patient who has a thick-walled cavity.

A report of one lobectomy and two pneumonectomies for the control of pulmonary hemorrhage, in the presence of thick-walled cavities, has been presented.

Twenty-one instances of fatal pulmonary hemorrhage have been reviewed. Seven patients might have been saved by pulmonary resection on the basis of current surgical considerations.

Medical, surgical, and anesthetic considerations in the patients with pulmonary tuberculosis and hemorrhage have been discussed.

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CLINICAL MANAGEMENT OF POSTTRAUMATIC RUPTURE OF THE THORACIC AORTA

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FOLLOWING severe nonpenetrating thoracic injury, the aorta may be torn, producing either a complete laceration with immediate exsanguination, or an incomplete tear of the vessel wall with delayed rupture or the subsequent formation of an aneurysm. Although reports of such lesions have appeared sporadically for many years,[1-9] only recently has there been much clinical interest devoted to them, due no doubt to their increasing frequency as accidents incident to high speed transportation become more common, and to advances in thoracic and vascular surgery which now afford a reasonable therapeutic approach to their clinical management.[10-20]

The purpose of this report is to describe 5 cases of thoracic aortic rupture due to nonpenetrating trauma, all of which were diagnosed clinically, and each of which represents one phase in the clinical course and management of this problem. On the basis of these and other reported experiences, a clinical approach to these lesions is suggested.

CASE REPORTS

Case 1.—T. C., a 42-year-old man, suffered severe injury to the chest in an automobile accident in 1940, from which he recovered without incident. Upon his entrance into the army in 1941, a roentgenogram disclosed a mass in the left chest (Fig. 1, *A*) which was present also in 1944 and 1948 (Fig. 1, *B*). Retrograde aortography in March, 1957, confirmed the presence of an aneurysm in the descending thoracic aorta immediately below the left subclavian artery (Fig. 1, *C*, and *D*) which has remained constant in size since first visualized in 1941. This patient is entirely asymptomatic and is being followed by roentgenograms of the chest at 6-month intervals.

Summary: This posttraumatic thoracic aortic aneurysm located immediately below the left subclavian artery has been followed without evidence of its progression for 16 years.

Case 2.—J. H. F., a 24-year-old man, was in an automobile accident in November, 1951. In 1953, upon discharge from the army, he was told that there was an abnormal shadow observed in the roentgenogram of his chest. He was first seen at the Denver VA Hospital in March, 1954, and has been followed at 6-month intervals ever since (Fig. 2). He remains asymptomatic and there is no evidence that the aneurysm has enlarged during this period.

Summary: This posttraumatic thoracic aneurysm was first observed 2 years after injury and raised the clinical problem of its resection versus the advisability of watchful waiting. The latter method was chosen and the lesion has remained unchanged for 4 years.

From the Department of Surgery, University of Colorado School of Medicine and Denver Veterans Administration Hospital.
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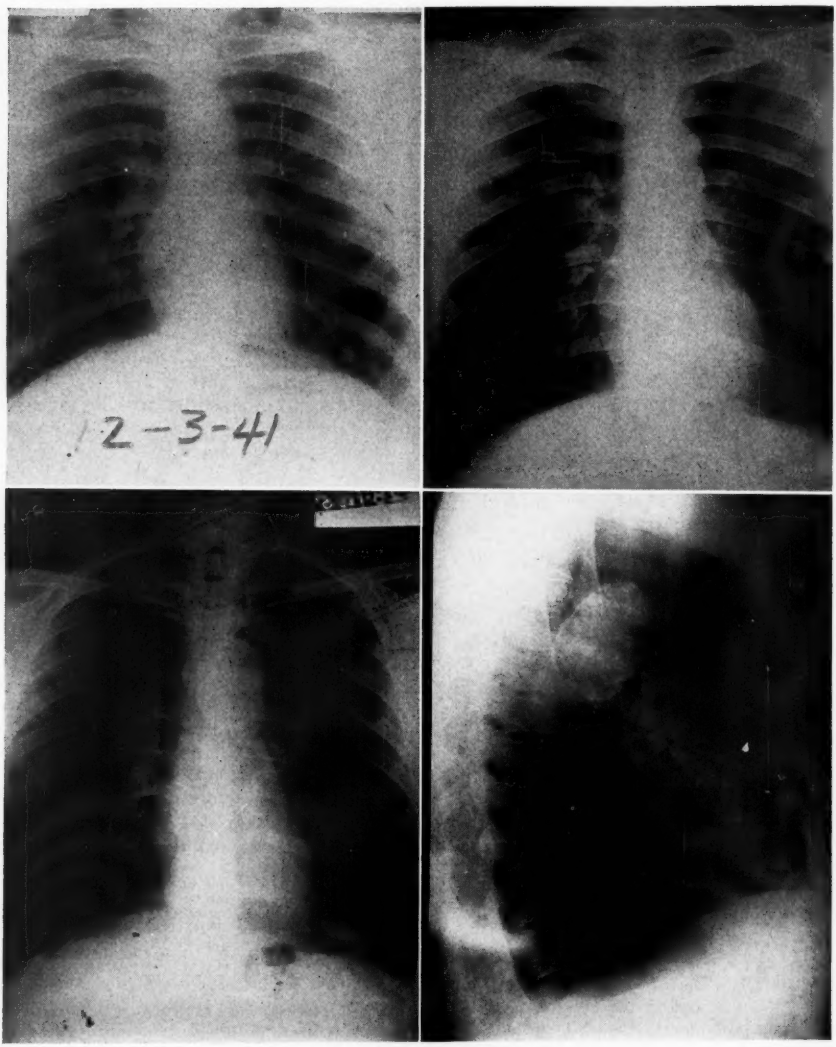
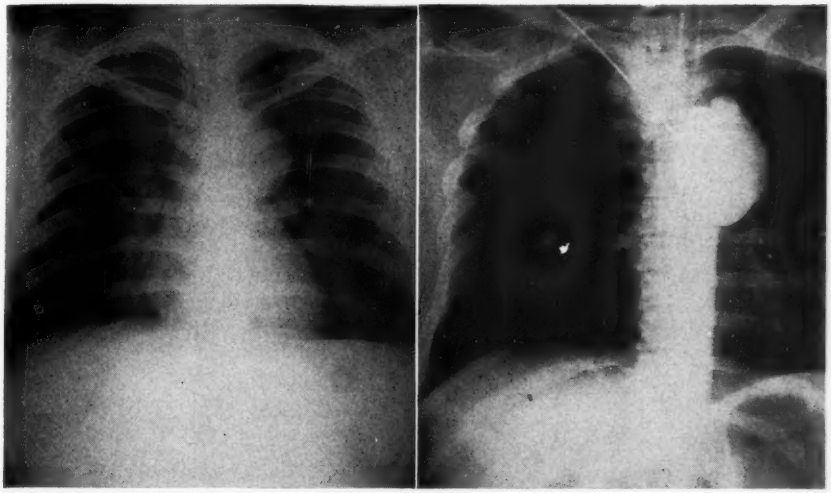


Fig. 1.—Case 1. *A*, (1941). One year following trauma showing slight widening of aortic arch.

B, (1948). Seven and one-half years folowing trauma. Aneurysm unchanged in size.

C and *D*, (1957). Posteroanterior and lateral chest x-rays 16 years following injury. No enlargement but marked calcification of the aneurysm.



A. *B.*

Fig. 2.—Case 2. *A*, (1954). Two and one-half years following injury showing aortic aneurysm. *B*, (1955). Transcervical aortogram showing aneurysm unchanged.

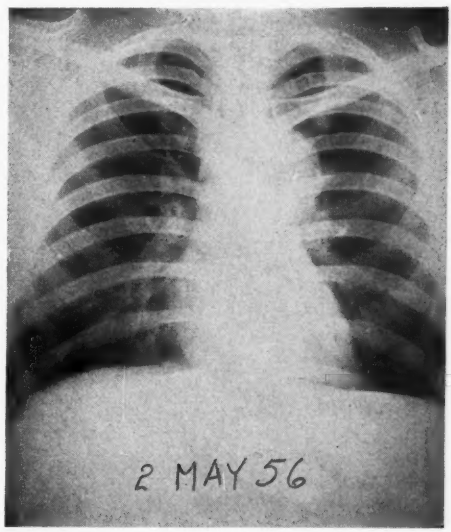


Fig. 2*C*.—Case 2 (1956). Four and one-half years following injury. Size of aneurysm unchanged.

CASE 3.—P. A., a 20-year-old man, suffered a fractured right olecranon and multiple lacerations of the face in an automobile accident on Jan. 1, 1957. He was hospitalized elsewhere and because of some anterior chest pain was thought to have a "possible fracture of the sternum." He was admitted to the Denver VA Hospital 2 days after injury, at which time his condition was good, although a roentgenogram of the chest (Fig. 3) showed questionable mediastinal widening and elevation of both diaphragms, patchy atelectasis, and minimal fluid in the left base. The olecranon fracture was repaired under general anesthesia without incident on the sixth postinjury day.

He was about to be discharged on the ninth postinjury day (Jan. 10, 1957), when he complained of moderate pain in his back and upper chest. His vital signs began to deteriorate later in the day, at which time breath sounds were found to be absent in the left chest.

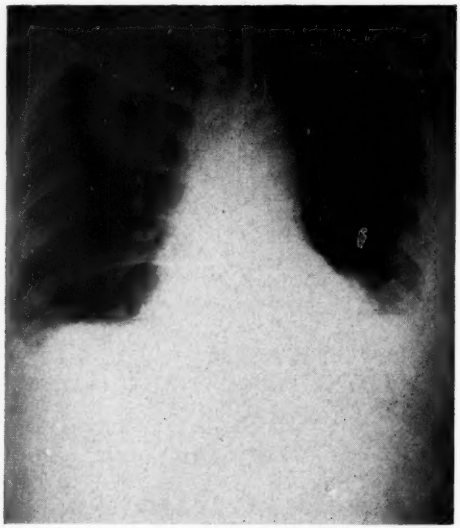


Fig. 3.—Case 3. Three days after trauma. Showing diffuse mediastinal widening and fluid at both bases but predominantly on the left.

Tubes were inserted into the chest, 1,000 c.c. of old blood removed, and his condition improved markedly. Two hours later his condition deteriorated, femoral pulses disappeared and he complained of numbness in his feet. As he was being taken to surgery (approximately 8 hours after his complaint of back pain) he suddenly went into shock. An emergency left thoracotomy revealed a ruptured thoracic aortic aneurysm. The bleeding was stopped by manual pressure over the torn aneurysm, and the heart resuscitated sufficiently to allow a formal thoracotomy. The aneurysm was resected and a nylon graft inserted, but the patient died shortly after the occluding aortic clamps were removed.

Summary: For 9 days following injury this patient had essentially no symptoms referable to the chest. Within an 8-hour period, however, he developed chest pain, hemothorax, and ultimately went into shock at the moment when the aneurysm ruptured into the left pleural space. Heroic operative measures were too late for subsequent recovery.

CASE 4.—R. V. A., a 44-year-old man, fell from a third story window during an epileptic seizure and struck his left chest on the ground, resulting in multiple rib and mandibular fractures. Upon entry to the Denver VA Hospital on Feb. 19, 1957, a roentgenogram of the chest showed a left pneumothorax and marked mediastinal widening (Fig. 4). The femoral pulses fluctuated in intensity but his general condition was satisfactory. A diagnosis

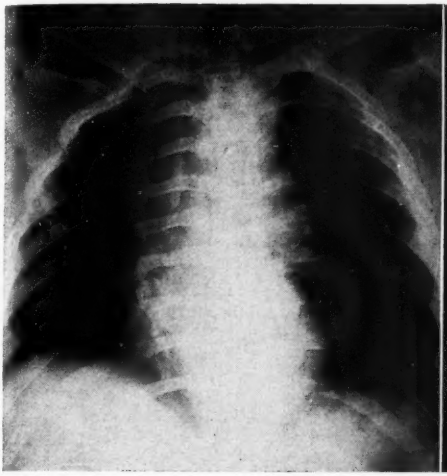


Fig. 4.

Fig. 5.

Fig. 4.—Case 4. Twelve hours after fall from third story window. There is marked mediastinal widening.

Fig. 5.—Case 4. Three weeks following injury. The angiocardiogram shows the aortic aneurysm that was not present at thoracotomy immediately following injury.

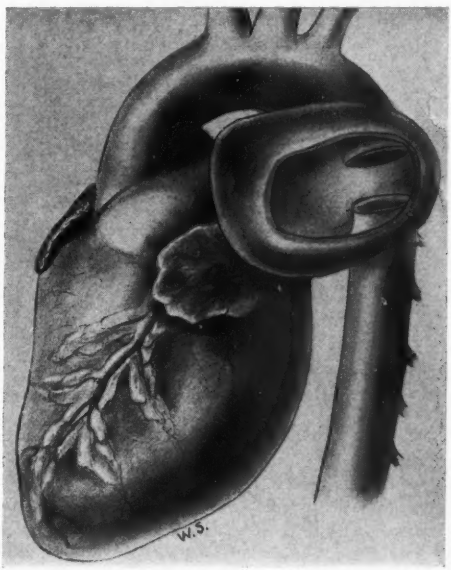


Fig. 6.—Case 4. Illustration of findings at second operation. The normal aortic wall has been circumferentially transected through the intima and media forming an aneurysm densely adherent to the left pulmonary artery, main-stem bronchus, and recurrent laryngeal nerve.

of thoracic aortic rupture was made and a left exploratory thoracotomy performed within a few hours after his admission to the hospital. Although there was diffuse ecchymosis surrounding the entire length of the thoracic aorta, no site of bleeding could be found. The aorta immediately below the left subclavian artery was minutely inspected, but no evidence of laceration was found. As the postoperative serosanguineous fluid finally cleared from the left chest, an aortic aneurysm was noted immediately beneath the left subclavian artery. This was first visualized on March 10 (Fig. 5), 21 days after injury and subsequent negative exploratory thoracotomy. Bronchograms the following day showed that the mass occluded the left upper lobe bronchus. At operation on April 3, 1957, under hypothermia anesthesia (29° C.), the aorta was cross-clamped below the left subclavian artery and the aneurysm opened. There was found to be a 360 degree circumferential, intimal tear with a 6 cm. separation of aortic wall opening into the aneurysmal sac (Fig. 6). The aortic wall above and below the aneurysm appeared normal. Twenty minutes after the aorta was clamped, as a lyophilized aortic graft was being inserted to bridge the defect, totally resistant ventricular fibrillation suddenly developed and the patient died.

Summary: Following severe injury to the chest, roentgenograms demonstrated widening of the mediastinum. Exploratory thoracotomy revealed diffuse periaortic hematoma, but despite close inspection of the aorta no evidence of rupture was found. Three weeks following injury and negative thoracotomy an aortic aneurysm was demonstrated beneath the left subclavian artery.

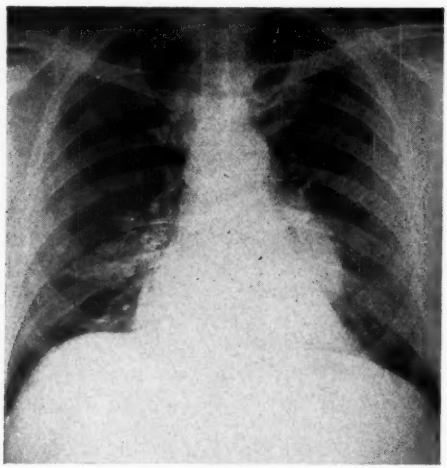
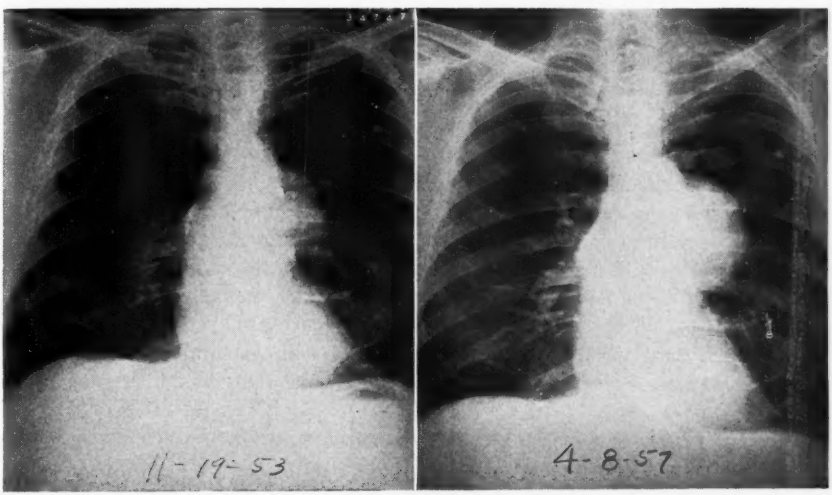


Fig. 7.—Case 5. Fifteen years after severe trauma showing aortic aneurysm involving the lower portion of the descending aorta. This lesion was successfully excised and replaced with a lyophilized homograft when it began to enlarge and cause symptoms.

CASE 5.—C. M., a 53-year old man, was in 2 automobile accidents, one in 1943 and another 1944. In both accidents he suffered severe injuries to the left chest and flank and had had broken ribs on this side. In 1953, a mass was noted in the left chest on a routine roentgenogram. He was asymptomatic, except for some low lumbar back pain, until 1956 when he began to notice pain in the left lower chest and back. X-ray examination of the chest (Fig. 7) showed a questionable enlargement of the mass compared to previous studies. May 21, 1956 (at the Weld County General Hospital, Greeley, Colo.), the patient was operated upon under hypothermic (28° C.) anesthesia and a lemon-shaped aneurysm overlying the seventh thoracic vertebra and measuring 11 cm. in greatest diameter was removed. A lyophilized aortic homograft was used for replacement. The patient's postoperative course was uneventful. Subsequently, the back pain disappeared, and he is gainfully employed.

Summary: Thirteen years following chest injury an aneurysm of the descending thoracic aorta was thought to be enlarging and producing back and chest pain localized to this region. It was successfully excised and replaced with a homograft, furnishing relief of symptoms.

CASE 6.—W. B. L., a 63-year-old man, suffered severe trauma to the left chest at the age of 17 when he was kicked by a mule. A mass was noted in the descending portion of the aortic arch in 1948, and roentgenograms of the chest showed gradual enlargement in 1953 (Fig. 8, *A*). He was first seen at the Denver VA Hospital in 1957, at which time the aortic aneurysm had appreciably enlarged (Fig. 8, *B*). Repeated analyses of the blood and cerebrospinal fluid were negative for syphilis. Because of advanced metastatic carcinoma of the bladder no direct attack on the aortic aneurysm has been advised.



A. *B.*

Fig. 8.—Case 6. *A*, (1953). Forty-three years following mule kick to left chest. *B*, (1957). Four years later and 47 years following injury showing marked growth in size of the aneurysm.

Summary: This descending aortic aneurysm is most likely due to severe trauma to the chest suffered 47 years ago. Proof of such etiology is, however, lacking. It has been followed by serial roentgenograms of the chest for 9 years and has shown marked enlargement during the past 4 years (43 years following injury).

The salient features of these 6 cases are summarized in Table I.

DISCUSSION

The number of cases of posttraumatic thoracic aortic aneurysms that have now been reported removes this lesion from the realm of curiosity to one whose natural history should be carefully described and its proper clinical management thereby delineated. The characteristic trauma producing these lesions and the hydrostatic principles involved have been well described elsewhere[20, 21] and need not be mentioned further.

TABLE I. SUMMARY OF CASES IN THIS SERIES

PATIENT	TIME INTERVAL INJURY TO DIAGNOSIS	REMARKS
J. H. F.	3½ years	No surgery; 5½ year follow-up; no increase in size of aneurysm
P. A.	10 days	Emergency surgery; excision with attempted graft replacement; died
R. Van A.	16 hours	Immediate thoracotomy with essentially negative findings; surgery 40 days posttrauma for aneurysm; attempted excision; death from ventricular fibrillation
T. C.	16½ years	No surgery; calcification of aneurysm but no increase in size
C. M.	13 years	Surgery with excision and homograft replacement; hypothermia; no neurologic sequelae
W. B. L.	43 years	Followed for 9 years with marked enlargement of aneurysm for last 4 years; no surgery because of inoperable bladder carcinoma

Natural History of the Lesion.—Complete laceration of all layers of the aorta by nonpenetrating chest trauma follows extremely severe injury and results in almost instantaneously fatal exsanguination. Although such lesions are common findings at the coroner's inquest following airplane accidents,[21] they have little significance for the clinician. In the future the proper use of seat belts and other devices may reduce the number of such immediately fatal injuries.

Following incomplete severance of the aortic wall, on the other hand, there is a varying period of time before delayed perforation may develop or before an aneurysm may form. Their proper clinical management depends on the developmental course of the untreated lesion, which heretofore has not been emphasized.

More than one half of the aortic tears following blunt thoracic trauma are located immediately below the left subclavian artery,[6] the remaining being either at the origin of the aorta from the heart, on the concavity of the arch, or in the descending aorta. Commonly the tears are transverse and occasionally are completely circumferential. The intima is torn but with a varying thickness of the adventitia and media remaining intact. External examination of such lesions immediately following injury reveals (Case 4) only a diffuse subadventitial ecchymosis along the aorta, without any localizing collections of blood or bulging of the vessel wall.

The time that the unbuttressed aortic adventitia can withstand arterial pressure before it ruptures, or an aneurysmal dilation occurs, undoubtedly depends upon the depth and extent of the original internal laceration. The adventitia accounts for 60 per cent of the tensile strength of an artery[22] but will ultimately give way if the other layers are disrupted. The significant fact is that such a latent period exists following injury, during which time the lesion may be overlooked by the unsuspecting clinician. On the other hand, if the progressive nature of the lesion is recognized, an aggressive surgical approach during this period may be of clinical benefit in avoiding aortic rupture. The time between injury and aortic rupture in Case 3 was 9 days. The previously reported experience in this regard is summarized in Table II and illustrates that

TABLE II. SUMMARY OF CASES PREVIOUSLY REPORTED

AUTHOR	NO. CASES	TIME FROM INJURY TO DIAGNOSIS	REMARKS
Kemp (1923)[1]	1	Immediate	Tear, ascending aorta; post-mortem diagnosis
Kuhn (1925)[2]	1	10 days	Post-mortem diagnosis
Corbitt and Mathews (1937)[3]	1	20 hours	Post-mortem diagnosis
Kleinsasser (1943)[4]	1	Immediate	Post-mortem diagnosis
Forbes (1944)[5]	1	30 hours	Post-mortem diagnosis
Strassman (1947)[6]	69	1-18 hours	Review of 7,000 autopsies
Henning and Agmar (1948)[7]	1	48 hours	Post-mortem diagnosis
Bradford and Johnston (1950)[8]	1	5 weeks	90 cases reviewed; 7 survived over 18 hours
Rice and Wittstruck (1951)[9]	1	20 days	Female at term pregnancy
Hollingsworth et al. (1952)[10]	4	4 months 2 years, 4 months 1 year 2 months	1-7 year follow-up; 2 cases with thoracotomy only; 1 case had Cellophane wrapping of aneurysm
Wyman (1953)[11]	3	8 days 15 hours 11 hours	Post-mortem diagnoses; all were aneurysms of posterior aortic arch
Kastl (1953)[12]	1	10 days	Post-mortem diagnosis
Bahnson (1953)[13]	1	8 months	Saccular aneurysm; excision with closure
Goyette et al. (1954)[14]	3	2 years 8 years 4 months	Thoracotomy; no attempted repair; no surgery because of location; excision; died 3 months later from rupture
Bornstein (1954)[15]	1	4 hours, 10 minutes	Chest x-ray 3 hours posttrauma said to be negative; post-mortem diagnosis
Hardin et al. (1954)[16]	1	2 years	Increase in size over 8-year period; excision and graft; hypothermia; operative death from ventricular fibrillation
Stranahan et al. (1955)[17]	1	5 years	Successful resection and homograft replacement; temporary by-pass shunt
Sarot and Lazzarini (1955)[18]	1	14 years	Successful resection and homograft replacement; permanent by-pass shunt
Cooley and DeBakey (1955)[19]	1	8 years	Tangential aneurysmectomy; later excision and graft; successful
Zehnder (1956)[20]	1	4½ days	Post-mortem diagnosis

essentially all such episodes occur during the first 3 weeks following injury. Although there may be little or no clinical evidence of aortic damage during most of this latent period, a short premonitory period usually precedes perforation and exsanguination. Like its atherosclerotic and syphilitic counterparts the traumatized aorta characteristically begins to leak slowly at first, producing symptoms of hemothorax, respiratory distress secondary to collapse of the left lung, and evidence of progressive blood loss. This pre-exsanguination period is the surgeon's last chance to staunch the flow of blood from the crumbling aneurysm wall, which like a distintegrating dam may start with a trickle but soon ends in a disastrous flood. The period from the first signs of perforation to death may be but a few minutes or as long as 24 to 48 hours.

Early aortic rupture following trauma characteristically occurs within the first 3 to 4 weeks. Thereafter an anuerysm may develop which, appearing as they do in an otherwise normal aorta, have a better prognosis than their syphilitic or atherosclerotic counterparts. Whereas most of the latter rupture within 8 months after onset of symptoms,[23] there is increasing evidence that many posttraumatic aneurysms found one month or more after injury remain indefinitely unchanged.

Clinical Management.—An immediate free aortic perforation or transection presents no clinical problem inasmuch as the casualty seldom survives to receive even the most preliminary medical attention.

When an incomplete aortic tear occurs following nonpenetrating trauma the clinical problem resolves itself into: (1) is there actually an aortic injury, (2) will the aorta perforate, (3) is an aneurysm developing, and (4) if an aneurysm is present, is it expanding or static and should therefore be excised?

A high index of suspicion for aortic injuries following severe nonpenetrating trauma to the chest is essential for their early recognition. Numerous case reports in the literature, in which the diagnosis was unsuspected until postmortem examination, testify to the previous ignorance of this lesion. Once aortic damage is suspected, examination of the chest must be done to determine questionable widening of the mediastinal shadow and to delineate the outline of the aorta. Vital signs should be recorded at frequent intervals for evidence of major blood loss and suitable measures provided for replacement therapy. Progressive widening of the mediastinal shadow, signs of blood loss, or unexplained left hemothorax necessitate exploratory thoracotomy at the earliest feasible moment, while the patient's general condition remains satisfactory.

The large number of reported cases that have been clinically unsuspected and treated by nonoperative means for hours or even days following injury, while they bleed fatally into the chest, make early thoracotomy mandatory if there is serious possibility of aortic rupture.

An incision in the left fourth interspace will allow adequate exposure of the entire aorta and an optimal approach to the area immediately inferior to the subclavian artery where most of the lacerations will occur. If there is any evidence of periaortic ecchymosis (as in Case 4), the entire thoracic aorta must be both minutely inspected and palpated for evidence of an occult tear.

Should no such laceration be found to account for the periaortic hematoma, the question arises as to the advisability of an exploratory aortotomy performed between occluding clamps immediately below the subclavian artery so as to visualize the posterior wall of the aorta at this point. In the case cited, such a maneuver would in all likelihood have saved the patient's life.

A small incomplete aortic laceration found in the immediate posttrauma period, before aneurysmal dilatation has developed could probably be successfully repaired by simple vascular suture without the necessity for prolonged aortic occlusion with resection or grafting. Since such lacerations characteristically appear in the posterior aspect of the aorta, adequate exposure may be difficult.

The clinician must remain vigilant for signs of aortic rupture for at least 4 weeks following injury and must not ignore such a catastrophic possibility merely because of several asymptomatic days or even weeks. Normally there will be a warning period of a few hours, during which time the alert and aggressive surgeon will have time to perform thoracotomy before massive hemorrhage supervenes.

Finally, the clinician may be faced with the problem of an asymptomatic aneurysm discovered months or even years following injury. Since there is good evidence that some of these lesions may remain unchanged for many years, such aneurysms probably can safely be followed at 6-month intervals by serial roentgenograms or laminograms. Should there be incontrovertible evidence of enlargement, or should the lesion become symptomatic, excision and graft replacement can be employed.

Hypothermia (to 29 to 30° C.) and some by-pass shunt around the aneurysm should be used, both to minimize the likelihood of spinal cord damage and to relieve the heart of the burden of working against an occluded thoracic aorta. The hypothermic heart tolerates poorly such outflow resistance unless inflow occlusion is simultaneously employed.[24]

SUMMARY

1. Six clinical cases of posttraumatic thoracic aortic aneurysm have been described which represent various phases of the natural history of this condition.

2. On this basis and on the previous experience of others, the following clinical approach is suggested:

a. Early thoracotomy should be performed if incomplete aortic laceration is seriously suspected. Such a procedure should include careful inspection and palpation of the entire length and circumference of the thoracic aorta for evidence of laceration. Exploratory aortotomy may even be necessary to visualize the site of rupture if no other method discloses the origin of diffuse periaortic ecchymosis.

b. An asymptomatic period of 2 to 3 weeks may elapse following severe chest injury before delayed aortic rupture occurs.

c. Immediately prior to the exsanguinating hemorrhage from an incomplete aortic laceration or from the resulting aneurysm, there commonly is a short

(1 to 36 hour) period of a lesser leak. This premonitory warning must be recognized instantly and immediate thoracotomy performed for operative cure.

d. Asymptomatic posttraumatic thoracic aortic aneurysms discovered incidentally more than a month following injury probably will stabilize and can be managed expectantly with serial roentgenograms of the chest at 6-month intervals. Should there be any evidence of enlargement or development of symptoms they should be excised and replaced with a suitable graft.

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TRANSFIXION OF THE AORTA ACCOMPANIED BY A BROWN-SÉQUARD SYNDROME

A Case Report

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THERE are few reports of successful suturing of lacerations of the thoracic aorta and survival is rare. The first successful suturing of an aortic laceration was by Dshanelidze,[4] in 1922. His patient was a 20-year-old man with a stab wound to the left of the sternum in the second interspace. The pericardium was opened and an 8 mm. wound about one centimeter from the heart was closed. In America, Blalock[2] reported the first successful suture of a similar wound, in 1932. His patient was an 18-year-old man with an ice pick laceration through the first left interspace and the intrapericardial portion of the ascending aorta. In 1941, Elkin[5] reported 2 cases of ice pick perforation of the ascending aorta in which operation was performed with one survival. Again, in 1944,[6] he presented a case of a stab wound of the intrapericardial aorta with survival after suturing. Beattie and Greer[1] reported what they believed was the fifth successful repair of a lacerated thoracic aorta. Their patient, a 21-year-old man, had been stabbed in the second left interspace and forty-eight hours later a 6 cm. portion of knife blade was removed from the ascending aorta within the pericardium. The 5 reported successful cases all concerned the intrapericardial ascending aorta and were remarkably similar wounds.

Significantly, in 1950, Bradford and Johnston[3] reviewed the literature and reported 90 cases of traumatic rupture of the aorta with only 4 patients surviving the injury. All of these survivors had sustained the aortic tear within the pericardium. Worthy of mention are 2 recorded cases of arteriovenous fistula between the aortic arch and the left innominate vein following stab wounds. The first successful closure of such a fistula was by Proctor in 1950.[8] His patient was a 25-year-old railroad employee who was struck by flying glass just above the left sternoclavicular joint and sustained a minor laceration. He presented himself a year later with cardiac failure and "buzzing" in his head. An almost exact duplicate of this case was reported by McCook.[7] His patient was stabbed in the same location and developed an identical fistula which was successfully closed.

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Our case is unique, representing survival after successful closure of a perforating, transfixing stab wound of the descending thoracic aorta complicated by a Brown-Séquard syndrome due to concomitant spinal cord damage.

CASE REPORT

L. B., a 44-year-old prison inmate, entered the Hospital of the University of Pennsylvania, on Dec. 2, 1956, approximately twenty-seven hours after being stabbed in the back, chest, and left arm by a fellow prisoner. The previous medical history was not significant. The patient was given penicillin the day of injury and morphine for severe pain referred to the left hip and radiating over the distribution of the left sciatic nerve.

The physical examination revealed a well-developed, well-nourished, alert, 44-year-old Negro man in no acute distress. The vital signs were normal. There were multiple stab wounds of the left chest and arm, some of which were sutured. There was a puncture wound of the back about 2 cm. to the right of the midline at the level of the eleventh thoracic vertebra. The chest expanded equally and well and breath sounds were normal. The cardiac rhythm was regular and no murmurs were audible. The abdomen was soft and flat with normal peristalsis. Anal sphincter tone was good. All peripheral pulses were

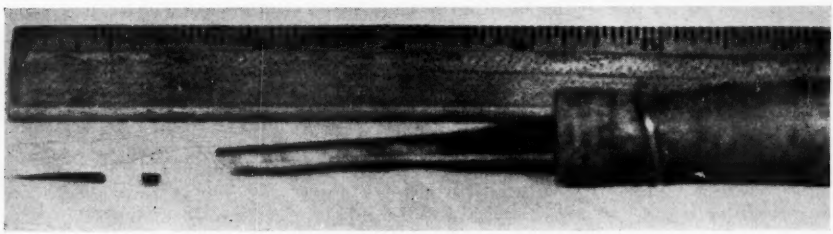

Fig. 1.—Stiletto and blade remnant with two pieces about one inch long removed from the anterior aspect of the eleventh thoracic vertebra.

palpable and equal. The neurological examination revealed all cranial nerves intact, a flaccid paralysis of the left lower extremity from below and including the hip girdle muscles with intact sensation, loss of pain and temperature sensation over the right lower extremity below the gluteal fold, absent deep tendon reflexes in the left knee and ankle, and diminished abdominal reflexes on the left.

The injuries were inflicted by a stiletto, ground from a steel file into a tapering, elliptical shape and affixed to a five-inch wooden handle (Fig. 1).

Studies revealed a hemoglobin of 16.5 Gm. and a leukocyte count of 12,700. Routine studies of blood chemistries were within normal limits. A lateral chest film showed a knife blade approximately 9 cm. long which had traversed the neural arch and body of the eleventh thoracic vertebra, the tip extending beyond the vertebral body and possibly transfixing the aorta. In the anteroposterior view, a metallic foreign body only 0.5 by 1.4 cm. was visible (Fig. 2). There was also a longitudinal fracture of the neural arch of the eleventh thoracic vertebra. No other abnormalities of the thoracic cage or contents were noted.

The patient was operated upon within two hours of admission. It seemed possible that the portion of the knife blade, protruding anterior to the body of the vertebra, transfixed the aorta. The remnant was to the left of midline and almost directly in a sagittal plane. It was elected to carry out a laminectomy over T_{10}, T_{11}, and part of T_{12} and, if the blade could be reached and removal deemed possible, thoracotomy would then be performed to safeguard against massive hemorrhage.

The patient was placed in the right lateral decubitus position and, under endotracheal anesthesia, a laminectomy over T_{10} and T_{11} was performed. Upon exposing the dura, no

tear was seen. It was then opened and the spinal cord found to be slightly edematous and congested. A No. 14 rubber catheter was passed upward and downward along the cord for a distance of three or four inches without encountering a block. The knife blade could not be found. A thoracotomy was then carried out through the ninth left interspace. The diaphragm was partially opened to facilitate exposure. The point of the knife was then easily palpated within the thoracic aorta at the level of the eleventh thoracic vertebra about ⅛ of an inch from the anterior wall in the midline of the vessel. A retropleural hematoma was present around the aorta posteriorly from the diaphragm to about the ninth thoracic vertebra and consisted of approximately 200 c.c. of blood. The aorta was then mobilized, occluded above and below the point of entrance with Crafoord clamps, and lifted off the knife blade anteriorly. The laceration was 1.5 cm. long and was closed with a continuous suture of No. 4-0 arterial silk. The time of total occlusion of the aorta was seven minutes. Various attempts to manipulate the protruding knife blade were unsuccessful since it was firmly imbedded in the vertebral body. Finally it was broken off

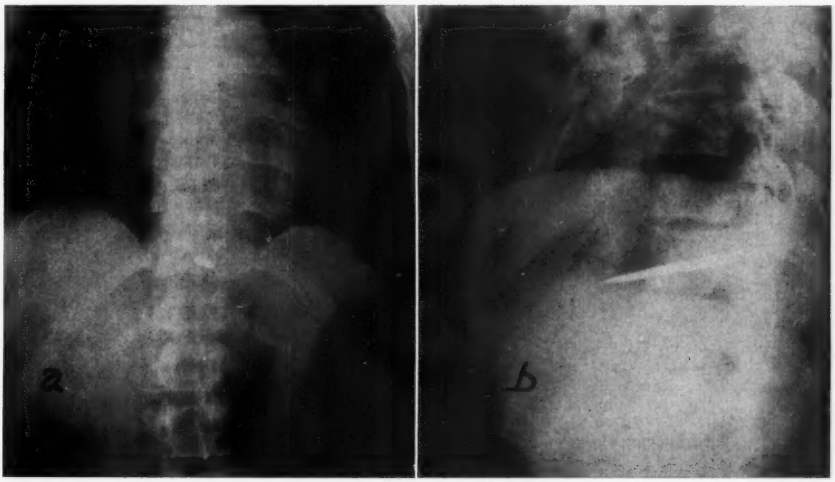

Fig. 2.—*a*, Anteroposterior view showing blade in sagittal plane to the left of midline and at the level of the eleventh thoracic vertebra.
b, Lateral view of lower chest and abdomen showing knife blade through the eleventh thoracic vertebra and protruding anteriorly about an inch.

flush with the vertebral body and the remainder left in situ. The removed portions are shown in Fig. 1. The chest was closed with a large drainage tube connected to a water-seal bottle. The patient received a total of 4,500 c.c. of blood during these procedures which he tolerated very well. Postoperative x-rays revealed that the laminectomy was one vertebra too high, but even so it is believed that the blade remained extradural.

Postoperatively the patient developed weakness of the right lower extremity and this gradually improved. The patient complained of moderately severe upper abdominal pain during the first one to two weeks of convalescence and had associated guarding and tenderness, probably on a reflex basis. Peristalsis was normal and the patient was able to eat without difficulty. On the seventh postoperative day, the patient exhibited a left Babinski reflex which indicated a beginning mass flexor withdrawal reflex and subsidence of spinal shock. This was significant prognostically since contusion and not laceration of the spinal cord had therefore occurred. The initial bladder catheter drainage was discontinued and the patient voided normally. He was discharged back to the prison on Dec. 12,

1956. A follow-up report two months later revealed that some atrophy of the left calf muscles had occurred but that considerable motor power had returned to the left lower extremity and pain sensation had returned to the right.

SUMMARY

The first known instance of successful closure of a stab wound of the extrapericardial thoracic aorta is reported. This represents the sixth known case of successful suturing of a thoracic aorta laceration. The descending thoracic aorta of a 44-year-old man was transfixed by a stiletto plunged through the body of the eleventh thoracic vertebra, contusing the spinal cord and producing a Brown-Séquard syndrome. The literature is briefly reviewed.

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BRONCHIAL CLOSURE WITH CATGUT SUTURES

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IN JULY of 1956, Björk[1] published a report on suture materials used to close divided main and lobe bronchi. Approximately 29 of his 224 patients, with bronchial closure using nonabsorbable suture materials, developed suture granulomas, whereas none did so when the closure was effected with chromic catgut. His incidence of bronchopleural fistula did not increase when chromic catgut was used. Björk also described the use of chromic catgut as early as 1929 by Brunn[2] for a one-stage lobectomy.

Although the diagnosis of endobronchial suture granuloma per se was not generally used at Glenn Dale Hospital, the bronchoscopic findings when a bronchopleural fistula could be visualized implicated the suture as an undesirable foreign body in the infected bronchial stump. In surgery of other parts of the body, silk stitches as well as other permanent suture materials that have interstices have long been known to be undesirable foreign body foci. These can and will make surgical infections chronic and lead to sinus formation. In addition to the granuloma effect of a foreign body plus infection, the possibility of a foreign body promoting and/or potentiating a bronchopleural fistula is implied. It is important to us to have a dependable method of bronchial stump closure with absorbable suture material to avoid the foreign body effects of nonabsorbable material.

In addition to the role of silk sutures in infections, it is the opinion of the medical staff at the Glenn Dale Hospital that some patients have been known to suffer from a chronic cough which abates after the expectoration of a silk stitch. This was occasionally associated with a bronchitis similar to those reported by Pellnitz.[3] An exact tabulation of these cases of stitch cough is not possible except for the few that had stitches removed by the bronchoscopist. Nevertheless, the clinical experience of physicians following hundreds of postoperative cases for many years cannot be overlooked. This experience was continued even after the advent of drug therapy, although the number of such cases was thought to be considerably less since the beginning of streptomycin therapy.

To gain the advantages of absorption of suture material in bronchial stump closure, we began a clinical trial of chromic catgut closure of the bronchus in September, 1956.

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METHODS AND MATERIALS

A total of 60 consecutive pulmonary resections for far advanced and moderately advanced tuberculosis in which all the bronchi were closed with chromic catgut were studied. As a control series, 60 consecutive pulmonary resections for surgically similar far advanced and moderately advanced tuberculosis in which all the bronchi were closed with silk stitches were compared with the chromic catgut cases. These control patients had had 60 similar types of pulmonary resections immediately preceding, in time, the catgut group. Two of the patients who had had silk closure had to be eliminated as they were surgically dissimilar by virtue of their having received intrathoracic injections of Varidase for hemothorax. Otherwise the consecutive order was rigidly followed. The methods of operation, postoperative care, and general management were not changed from group to group. For purposes of the study the pulmonary resections at the Glenn Dale Hospital were separated into 7 basic classes: pneumonectomy, 12 cases; bilobectomy, 5 cases; lobectomy, 15 cases; lobectomy together with simultaneous segmental resection, 7 cases; lobectomy with wedge resection, 6 cases; segmental resection alone, 9 cases; and finally, segmental resection together with wedge resection, 6 cases. The catgut closure cases were matched according to the type of procedure with consecutive silk closure cases as outlined in Table I, making a total of 120 patients studied.

TABLE I

	SILK	CATGUT
Total number of individual bronchi closed (main, lobe, or segment)	78	73
Total consecutive patients	60	60
Number of cases according to resection classes:		
Pneumonectomy	12	12
Bilobectomy	5	5
Lobectomy	15	15
Lobectomy and segmental	7	7
Lobectomy and wedge	6	6
Segmental resection	9	9
Segmental and wedge	6	6

In each case the actual number of bronchi sutured was tabulated. Only the main bronchus, the lobe bronchi, and the segmental bronchi were recorded. Very small bronchi, such as those in the intersegmental planes, were not involved in any special closure technique. The small difference of 5 bronchial closures between the groups is accounted for by the difference in bronchial anatomy on the right and the left sides, as well as whether or not the bilobectomy cases were done through the intermediate bronchus or as two separate lobes such as the upper and middle on the right.

All patients received vigorous antituberculous chemotherapy administered in this hospital until the lesions were considered to have reached maximum resolution by x-ray studies and clinical conference studies. In the silk closure cases there were 25 patients who had had a positive sputum for tubercle bacilli by smear or culture 6 months prior to operation, or the resected specimens

were found to be smear or culture positive. In the cases of catgut closure, 21 patients had has similarly positive sputum smears or cultures preoperatively, or the tubercle bacilli were recovered from their resected specimens. The tubercle bacilli from sputa were found resistant to streptomycin, INH, or PAS in 5 cases of silk closure, and in 3 cases where the closure was effected with chromic catgut. All patients were bronchoscoped prior to resection. No patients with acute or subacute bronchial tuberculosis or bronchitis were resected. Histologic study of the bronchi at the resection line from the resected specimens was carried out and found to confirm the preoperative endoscopic impressions.

METHODS OF BRONCHIAL CLOSURE

A simple, single row of interrupted chromic catgut sutures was used to close the bronchi. The chromic catgut was open market purchase of Ethicon manufacture. In both the control and the catgut series, the bronchi were transected and then closed with 3-0 silk or 2-0 chromic catgut on an Atraumatic needle. Occasionally, 4-0 chromic catgut was used when anatomically necessary. Each suture was placed approximately 5 mm. deep in the cut end of the bronchus. This type of stitch avoids the crowding of masses of suture material close to the cut tissues which tends to interfere with local blood supply. In addition, the use of excessive amounts of suture material would tend, in the cases of nonabsorbable silk, to enhance the possibility of granuloma formation. Undue tension on the suture line was avoided. An airtight seal was effected as evidenced by the usual under-saline solution test, with increased endotracheal pressure provided by the anesthetist.

BRONCHIAL COMPLICATIONS

Table II summarizes the bronchial complications of each group. There were 5 postoperative pneumothorax problems in the control group of silk closure. All these occurred from the main, lobar, or segmental bronchi. Exact diagnoses as to these locations were accomplished by the clinical course, the x-ray picture and endoscopy in the 2 pneumonectomy cases, by bronchoscopy and x-ray studies in the one lobectomy and the one bilobectomy case, and by bronchogram in the one segmental resection case. In the catgut series, the

TABLE II

	SILK	CATGUT
Bronchopleural fistula of main, lobe, or segmental bronchi	5	1
Air-leak fistula of intersegmental planes	0	2
Total number of postresectional pneumothorax problems	5	3

fistula in the one pneumonectomy case was diagnosed by the clinical course, x-ray studies, and by the obvious result after re-intubation. The 2 cases of air-leak fistula from the intersegmental plane in the catgut cases were diagnosed in one case by open thoracotomy and direct visualization at the time of thoracoplasty, and in the second case by the roentgenogram and the clinical course. This

latter patient showed a slowly increasing pneumothorax immediately after original extubation of chest catheters that were placed in the hemithorax at the time of the operation. Re-intubation immediately resulted in full re-expansion and resolution of this pneumothorax. There was no fever or cough.

Resistance of tubercle bacilli to streptomycin, PAS, and INH, occurred in 5 of 25 control cases, in which tubercle bacilli were recovered from the sputum or the resected specimen. Of these resistant cases, 4 patients developed bronchopleural fistulas. In the chromic catgut series, there were 3 patients with similarly resistant tubercle bacilli, in a total of 21 cases in which tubercle bacilli were recovered from sputum or from the resected specimen. None of these 3 patients developed bronchopleural fistulas. In all the resistant cases, the patients were treated nevertheless with PAS, INH, and streptomycin, as well as the other new antituberculous chemotherapeutic agents such as viomycin and cycloserine, as such drugs become available. In 3 cases of resistant tubercle bacilli in which tuberculous bronchopleural fistulas developed, the bronchial stumps were visualized. In these instances, silk stitches were seen to be present and acting as centrally located foreign bodies surrounded by white and necrotic bronchial tissue. Removal of these stitches, when it was possible with the bronchoscope, seemed to aid the control of the tuberculous bronchitis. However, there was no dramatic cure of the tuberculosis in these patients with established tuberculous bronchitis when these stitches were removed. This study should not be construed to imply that such would be the case. Nevertheless the presence of these foreign bodies seemed deleterious and the pathogenesis of infection aggravated by foreign body was implied. One patient who had had segmental resection developed a nontuberculous silk stitch granuloma in a bronchial suture line, and was relieved of symptoms of cough and hemoptysis when the silk suture was removed and antibiotics were administered.

CONCLUSIONS

Sixty tuberculous patients had various types of pulmonary resections performed with 73 separate bronchi closed with chromic catgut. They had a satisfactory clinical course. Compared with the previous 60 surgically similar tuberculous patients who had 78 bronchi closed with silk, the 60 patients in the catgut series showed no increase of bronchial complications. In fact, there were 5 major bronchopleural fistulas in the silk group and only 1 major bronchopleural fistula in the catgut series. There were 5 resistant positive cases in the silk group, in which 4 tuberculous bronchopleural fistulas developed. There were 3 resistant positive cases in the catgut group and none developed bronchopleural fistulas. It is obviously impossible to follow the healing of bronchial stumps in patients by serial histologic studies. Further, it is recognized that the number of resistant cases is small and only possible trends can be recognized. Certain conclusions, however, have to be reached on the basis of clinical analysis and these are as follows: (1) Chromic catgut is as good as silk in effecting successful bronchial closure in resections for tuberculosis, whether or not the bacilli are resistant or sensitive to the usual antituberculous chemotherapeutic

drugs. This, in general, is in agreement with Björk's[1] findings. (2) The number of bronchopleural fistulas of the bronchi closed with catgut was lower than the number of bronchopleural fistulas in the cases closed with silk. The commonly known high frequency of bronchopleural fistula in resistant positive cases held true in the silk group in 4 cases out of 5, whereas 3 resistant positive cases in which catgut was used healed well. The suture material was the only change in the method of treatment of these 2 consecutive groups of patients. These data, together with the known effect of foreign bodies in infected areas, suggest that the absorbable material is more desirable than the silk material. Further clinical study of the resistant positive cases in relation to use of absorbable suture material is indicated. (3) In the years following resection the patients are likely to benefit from catgut closure because the possibility of sutures being coughed up or causing streaking or hemoptysis from the granuloma effect is unlikely when absorbable suture materials are used.

SUMMARY

Closure of bronchial stumps by chromic catgut in pulmonary resections for tuberculosis has been found to be quite satisfactory and consistently good. Sixty cases of 73 bronchi closed with catgut were compared with a control series of 60 surgically similar cases in which 78 bronchi were closed with silk. Catgut closure appeared to be superior to silk closure. The resistance of tubercle bacilli at time of surgery and the bronchial complications were discussed.

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THE KINKED BRONCHUS SIMULATING A BRONCHIAL STENOSIS

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IN CASES of pulmonary tuberculosis with atelectasis of the lung and a tuberculous empyema or a fibrothorax, an extrapleural resection is usually performed. During the performance of such an extrapleural resection it is often easier to complete the operation if, during an early stage, the bronchus, the artery, and the veins are divided. This practice has only been followed in cases where the pulmonary tissue is obviously destroyed by multiple cavities. In all other cases, I strongly advocate that decortication be performed first in order to evaluate how much lung should be removed. Following this principle has made it possible to save functioning lung in many cases. In one instance the right lower and middle lobes could be saved after having been completely atelectatic under a tuberculous empyema for eleven years. One year later 33 per cent of the total oxygen uptake occurred on the right side in this patient now found negative by guinea pig test. Even patients coming to surgery under the diagnosis of a bronchial stenosis with atelectasis and fibrothorax should not be routinely treated by resection. The aim of this paper is to advocate a partial decortication with thorough examination of the bronchus before any hilar structure is divided. Following this principle it was possible to save pulmonary parenchyma in 2 cases with a preoperative diagnosis of bronchial stenosis. The stenosis was found to be due to kinking of the bronchus after dislocation and fixation of the lung by pleural effusion, and fibrous scarring.

CASE REPORTS

CASE 1.—A 54-year-old woman developed left-sided pulmonary tuberculosis in 1929. An artificial pneumothorax was instituted and continued until 1932. Her pulmonary tuberculosis was then considered healed and x-ray studies of the lung remained unchanged for twenty-four years. A high fever then occurred and new x-ray studies showed that the left lung was completely atelectatic with a massive pleural effusion (Fig. 1). Bronchoscopy showed a concentric stenosis of the left main bronchus, similar to an old tuberculous stricture, 3 cm. below the carina. Bronchography was performed to determine the possibility of bronchial resection with bronchotracheal anastomosis but a complete obliteration of the left main bronchus, 2 to 3 cm. below the carina was demonstrated. An extrapleural pneumonectomy was planned. At operation, a fluid-containing fibrotic pleural sac was found and liberated from the chest wall and diaphragm. A partial decortication was made and the lung found better than expected. The decortication was completed and now the lung could be filled with air under high pressure. Some small and hard nodules were palpated in the superior segment of the left lower lobe but there was no indication for resection. The left main bronchus was dissected free in its entire length and a longitudinal incision made. The stenosis, diagnosed by bronchoscopy and bronchography, could not be

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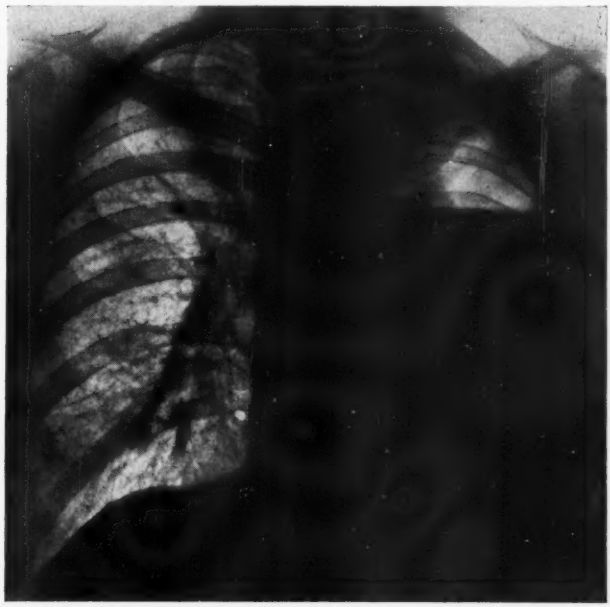

Fig. 1.—Case 1. Roentgenogram showing complete atelectasis of the left lung with a massive effusion.

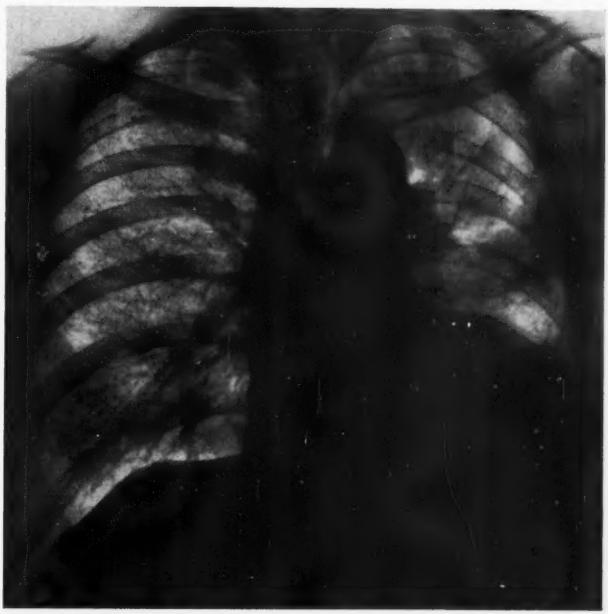

Fig. 2.—Case 1. Roentgenogram made three months after operation shows good aeration of the left lung and a wide open left main bronchus.

found, but the mechanism of bronchial obstruction could be reconstructed. As a result of the dislocation of the lung, the left main bronchus was kinked. After liberation and decortication of the lung, the obstructing kink disappeared. The bronchial incision was closed by interrupted sutures of No. 3-0 Chromic catgut. The patient madé an uneventful recovery apart from an atelectasis necessitating a bronchoscopy. Postoperative roentgenograms then showed good aeration of the left lung (Fig. 2).

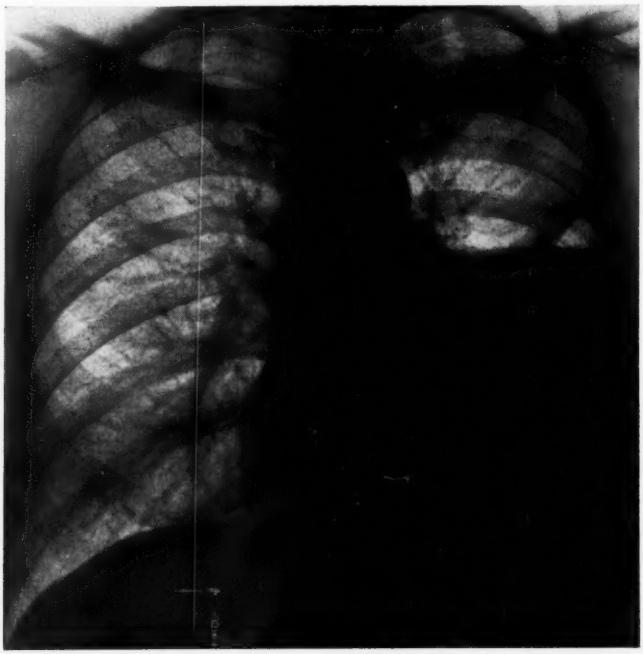

Fig. 3.—Case 2. Roentgenogram showing left lower lobe atelectasis with intrapleural effusion.

CASE 2.—A 58-year-old man had a left pleurisy in April, 1952. Guinea pig tests of sputum and pleural fluid were positive. Bronchoscopy showed a concentric stenosis of the left lower lobe bronchus. A tumor was suspected on two occasions, but biopsy was negative. On a preoperative diagnosis of left lower lobe atelectasis, due to a tuberculous bronchial stenosis or to a carcinoma (Fig. 3), an extrapleural lobectomy or pneumonectomy was undertaken. At operation (Oct. 24, 1952) the left lung was first completely freed by extrapleural dissection and decorticated. It was necessary to remove the visceral pleura over the lower lobe. After extirpation of the pleural sac, the left lower lobe was aerated. The left main and lower lobe bronchus was dissected free and found to be thin-walled and normal. As it was easy to palpate the normal wall of the lower lobe bronchus, having a 1 cm. internal diameter, this was not opened. There was no stenosis of the left lower lobe bronchus but the obstruction had been caused by kinking of the bronchus due to the fluid collection and fibrosis of the pleura. Immediately after the operation, bronchoscopy was performed and showed a normal left lower lobe bronchus. After removal of the tubes, an air pocket was formed which needed additional drainage for five days. Otherwise the recovery was uneventful and two months after the operation 32 per cent of the total oxygen uptake occurred in the left lung. In this case a planned lobectomy for bronchial stenosis ended up in a pleurectomy for a tuberculous pleurisy (Fig. 4).

SUMMARY

In pulmonary tuberculosis, bronchial stenosis is a common finding. Of 151 pneumonectomies for tuberculosis, 37 cases (25 per cent) were performed for stenosis of the main bronchus and in 31 cases the stenosis was on the left side.

In a series of 1,000 resections for pulmonary tuberculosis, 2 cases with a diagnosis of bronchial stenosis were found to have a kinked bronchus secondary to a dislocation of the lung due to pleural effusion and fibrosis. The cases illustrate the advantage of (a) decortication and inspection of the lung before division of the hilar structures, and (b) a longitudinal bronchotomy for verification or exclusion of a bronchial stenosis before the bronchus is divided in cases where the external appearance of the bronchus is not typical of a stenosis.

In 2 other cases a preoperatively diagnosed bronchial stenosis was, at operation, found to be due to kinking of the bronchus, but the lung was destroyed and had to be removed.

Fig. 4.—Case 2. Roentgenogram after decortication shows good aeration of the left lung.

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BRONCHIAL ADENOMA TREATED BY SLEEVE RESECTION OF THE MAIN-STEM BRONCHUS

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ALTHOUGH bronchial adenomas usually do not metastasize, they are locally invasive and must be considered potentially malignant tumors to be treated by wide local excision. When they involve the main-stem bronchus, adequate wide excision has generally meant pneumonectomy. In recent years, however, a number of cases have been reported in which the employment of bronchial anastomosis has resulted in the cure of main-stem bronchial adenomas by lobectomy rather than pneumonectomy. Only a few cases of local excision of the tumor and of portions of the bronchial wall with reconstruction of the bronchial tree without loss of lung tissue have been reported.

D'Abreu and MacHale[1] report the case of a 48-year-old woman with an adenoma of the left main-stem bronchus just below the opening of the left upper lobe bronchus which was treated by excising the entire thickness of the bronchial wall containing the tumor, leaving a minute strip on the anteromedial wall. The repair was by an end-to-end anastomosis using linen thread sutures. A follow-up bronchoscopy 25 months after operation showed the lumen at the anastomotic site to be two thirds of normal size.

Crafoord[2] cites the case of a young man in whom an adenoma infiltrated a part of the bronchial wall from just above the right upper lobe bronchus down to below the middle lobe bronchus. At thoracotomy, it was possible to do a radical local excision of the extrabronchial plum-sized part of the tumor, the infiltrated part of the bronchial wall, and the intrabronchial tumor with subsequent plastic repair of the bronchus. There was a tendency to stricture formation at the site of the bronchial repair which has made bronchoscopic dilatation necessary. Crafoord states that Price Thomas had a case which he successfully treated in the same manner.

Björk,[3] from Crafoord's clinic, states that in most cases of mucous and salivary gland tumors (adenoma and cylindroma) it is still necessary to perform a lobectomy due to long-standing bronchial obstruction causing bronchiectasis. But in early cases, when the changes behind the tumor are reversible a lung-sparing course is followed. He recommends that most of the intrabronchial portion of the tumor be electrocoagulated, giving a free air passage with re-expansion of the atelectasis and drainage of accumulated secretions. Some weeks later a block removal of the intra- and extrabronchial part of the

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tumor together with the infiltrated part of the bronchial wall should be carried out. He also states that the scar of a bronchial suture line has a great tendency to shrink causing a stricture, and making bronchoscopic dilatation necessary.

Goldman[4] reports the case of a 45-year-old woman with a carcinoid adenomatous polypoid tumor of the bronchus which recurred 5 years after the entire tumor was thought to have been removed through a bronchoscope. He found that it involved two rings of the right main-stem bronchus just distal to the carina. He had trouble controlling the proximal bronchus and used traction sutures on the carinal cartilage. An encircling bronchial excision was made rather than a sleeve resection and the continuity of the bronchus was restored without loss of lung tissue.

Gebauer[5] cites a remarkable case of Officer Brown of Melbourne in which the patient had an adenoma in the distal left main bronchus, opposite the upper lobe orifice, which obstructed the lower lobe bronchus and caused expiratory emphysema of the left lung. The main-stem bronchus was transected above the tumor, the upper lobe bronchus lateral to the tumor, the superior segment lower lobe bronchus posterior to the tumor and the basal bronchi transected below the tumor. The distal upper lobe stump was then sutured to the anterior edge of the basal stump and the superior segment to the posterior edge. Thus, the three divided bronchi were converted to a single stump which was anastomosed to the proximal stump of the main-stem bronchus.

These cases represent successful attempts at preserving the whole of the functioning lung by removing the local tumor along with varying portions of the underlining bronchial wall.

We recently encountered a bronchial adenoma originating in the left main-stem bronchus which we believe is the first such case reported in which an entire sleeve of the main-stem bronchus was excised with end-to-end restoration of the bronchus.

CASE REPORT

L. M., No. 15755-56, a 19-year-old white male had always enjoyed good health until 2 months prior to admission when he became aware of a short sporadic nonproductive cough. This persisted and one month prior to admission (March 23, 1956) he became aware of the gradual onset of shortness of breath. During this time he lost no weight, was not febrile, had no night sweats, no hemoptysis, and raised no sputum.

Three days prior to admission he developed pain in the left posterior chest. It was not pleuritic but aching in nature, and persisted to admission. Just prior to admission he had two small episodes of hemoptysis. A routine chest film in February, 1956, was interpreted as normal. On questioning, it was learned that the patient was very fond of eating peanuts.

Physical examination disclosed a temperature of 101° F.; a pulse of 120; respirations of 32; a blood pressure of 132/50 mm. Hg. Examination of the chest showed definite lag in respiration on the left; diminished percussion note, diminished tactile and vocal fremitus, and diminished to absent breath sounds over the lower two thirds of the left chest. Examination of the right lung was normal.

A roentgenogram (Fig. 1) disclosed an elevated left diaphragm with shift of the mediastinum to the left and some collapse of the left lower lobe. The clinical impression was aspiration of foreign body (peanut) or bronchial adenoma. Tomography did not reveal

any bronchial pathology. Bronchoscopy was performed three times. On the first two occasions massive bleeding was encountered as soon as the bronchoscope was introduced into the left main-stem bronchus and the examination had to be abandoned. On the third attempt a whitish friable tumor was seen about 2 cm. beyond the carina. Biopsy revealed adenoma of the bronchus. The tumor appeared to fill the entire lumen of the bronchus. From the anatomic location of the growth it was felt that a pneumonectomy would probably be necessary to provide an adequate resection.

Operation.—On April 27, 1956, under general intratracheal anesthesia, the left chest was opened through a posterolateral incision resecting the fifth rib and dividing the fourth and sixth ribs posteriorly for more adequate exposure. At operation, no collapse of the left lung was noted, but rather it was distended and firm and could not be compressed by manual pressure. It was obvious that a main-stem bronchial obstruction was present. Palpation of the entire lung and its hilum did not reveal any masses. The left main-stem bronchus was freed from the carina to the left upper lobe takeoff and, by palpating the bronchus, an olive-sized mass could be felt through its wall just beyond the carina in the

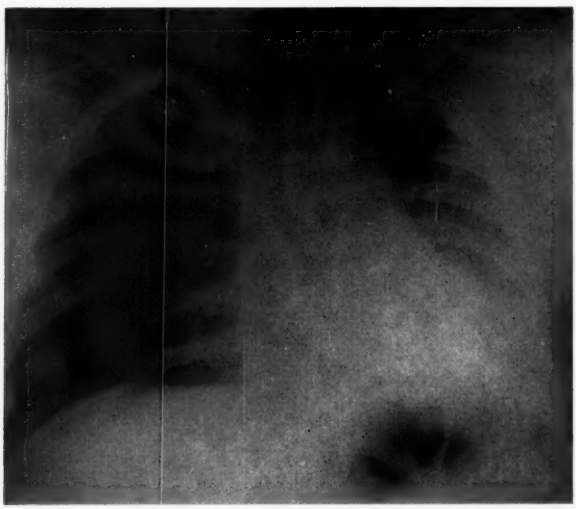

Fig. 1.—Admission film showing shift of mediastinum to left with interpretation of atelectasis of left lower lobe.

main-stem bronchus. It could not be visualized from the outside of the bronchus. It was elected to open the bronchus to delineate the pathology and visualize the problem. A 3 cm. longitudinal opening was made through the lateral wall of the main-stem bronchus. At first the thought of a local excision across the pedicle with cauterization of the base was considered but the base of the tumor was relatively extensive and such a course could not be defined as an adequate local excision. It was obvious that a generous local excision would necessitate the removal of a complete sleeve of at least 3 cm. of the left main-stem bronchus.

It should be mentioned here that, when the bronchotomy was first made, enormous quantities of thick white mucoid material extruded from the distal bronchus. The lung was then completely decompressed by squeezing it with extrusion of the above described material from the distal cut bronchus. Culture was taken of this material. Although its appearance and the clinical febrile course of the patient suggested massive infection of the left lung, it was elected to try to save the lung realizing that if the lung was irreversibly damaged a pneumonectomy might be necessary later on.

Before the bronchus was transected it was realized that some provision had to be made to prevent the proximal bronchus and carina from retracting into the mediastinum out of the operative field. Retention sutures would probably not hold the marked tension necessary to bring the carina into view and, in addition, would have been in our way. Therefore, by blunt dissection with the fingers, the trachea just above the carina was freed and, after an intratracheal catheter was passed beyond the carina into the right main-stem bronchus, a tape was passed around the trachea and tension was placed on it so the proximal edge of our resection was brought well into the operative field (Fig. 2). A complete sleeve of the main-stem bronchus comprising 3 cm. from just below the carina to just proximal to the left upper lobe take-off was excised and without tension the two ends were brought together using interrupted 3-0 silk sutures. Following this reconstruction the lung expanded and collapsed quite well and the suture line retracted into the mediastinum. Although the fate of the lung was still uncertain, it seemed that it had a reasonable chance of surviving. The chest was closed using two chest tubes for suction drainage.

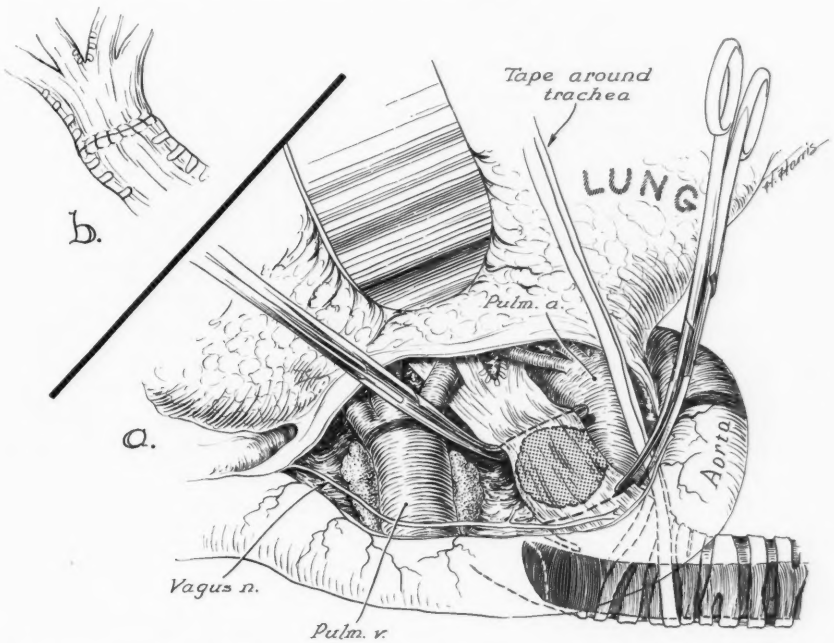

Fig. 2.—*a*, Operative field showing adenoma in left main-stem bronchus. *b*, Completed end-to-end anastomosis after sleeve resection of the bronchus.

Immediately following the operation, the patient's temperature returned to normal from the preoperative level of 101° F. The patient's postoperative course was completely uneventful. Roentgenograms of the chest showed that the mediastinal shift to the left returned to the midline and the left diaphragm again functioned normally (Fig. 3). The creamy material that extruded from the lung after bronchotomy was sterile on culture. Bronchoscopy, three weeks after operation, disclosed a patent left main-stem bronchus with some irregularity of the anastomotic site. The silk sutures could be visualized. Bronchoscopy, 8 months after operation, disclosed a completely full-sized left main-stem opening with no sutures or other evidence of a visible anastomosis. The bronchoscope was easily

passed through the area of the anastomosis into both the left upper and left lower lobes of the lung. A postoperative bronchogram revealed a normal appearing left bronchial tree without evidence of bronchiectasis.

Comment: This case represents one which, we had expected would require a pneumonectomy but was successfully treated by a sleeve resection of the main-stem bronchus.

Fig. 3.—Postoperative film showing clear left lung field and mediastinum returned to the midline.

DISCUSSION

We believe this case represents the first complete sleeve resection of a main-stem bronchus with successful reconstruction for adenoma of the bronchus. This is not technically difficult if the trachea above the carina is mobilized and pulled downward by an encircling tape. The problem of postoperative stenosis or stricture formation is insignificant if a complete sleeve of bronchus is removed rather than leaving a bridge of tissue on one wall. Where a sleeve is taken the anastomosis is even and symmetrical without the puckering or distortion that results in stenosis or stricture formation. This patient's symptoms and admission x-ray studies suggest that the left main-stem bronchus was blocked completely for from 2 to 3 months, yet the cultures were sterile at operation and no bronchiectasis was found by bronchogram in the blocked lung. Thus, it appears that fairly long-standing complete bronchial obstruction does not necessarily produce any irreversible damage to the lung, providing infection is not present. Such local resections, of course, predicate an adequate margin of resection lest the tumor recur at the anastomotic site as these neoplasms are prone to do if completely removed.

Although a completely intraluminal bronchial adenoma lying within the main-stem bronchus must be a relatively rare anatomic finding in adenomas

of the bronchus and lung, this case demonstrates what can be done with bronchoplastic techniques and a plea is made to explore such a course before resorting to the usual pneumonectomy in these cases.

SUMMARY

1. Bronchial adenomas are potentially malignant tumors and must be treated by wide local resection.

2. When adenomas occur in the main-stem bronchus, pneumonectomy has been the procedure of choice.

3. A case of an adenoma lying within and completely obstructing the left main-stem bronchus in a 19-year-old male is presented.

4. A wide local resection was performed by removing a 3 cm. complete sleeve of the bronchus with end-to-end anastomosis of the bronchus and no sacrifice of lung tissue.

5. The anastomosis was made possible technically by mobilization and traction of the trachea above the carina.

6. Although the left main-stem bronchus had apparently been totally blocked for several months, the retained secretions were sterile and no irreversible damage of the lung could be demonstrated.

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PRIMARY EXTRAGENITAL CHORIOEPITHELIOMA IN THE MALE MEDIASTINUM

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PRIMARY extragenital, mediastinal chorioepithelioma, although uncommon, is strangely enough a disease peculiar to men. The first proved case in a female was reported in 1952.[1] Although first described in the male mediastinum by Ritchie,[32] in 1903, mediastinal chorioepithelioma usually represents metastasis from a testicular cancer. Most authors maintain that every case must have a meticulous serial block section of the testes before an extragenital primary chorioepithelioma can be diagnosed,[2-5] and that any case showing small cysts or scars in the testes must be discarded, since they may represent primary testicular tumors undergoing involution.[4, 6, 7, 11]

Others require less stringent criteria, believing careful gross examination and representative sectioning sufficient.[8, 9] It is reasoned that small cysts or scars do not necessarily represent a tumor, but may represent the reciprocal effect of the extragenital hormone production. It is likewise pointed out in relation to mediastinal primaries, that testicular tumors seldom show anterior mediastinal deposits. In one series of 220 cases of metastasizing testicular tumors, none showed anterior mediastinal spread.[8] Some authors[10] question the validity of spontaneous healing of any testicular tumor. Thus, the valid number of reported cases of extragenital chorioepithelioma in the male, though small, will vary with criteria. This is further confused by the microscopic picture, terminology, and origin of extragenital chorioepithelioma in the male.

Up to 1957, 24 accepted cases of extragenital chorioepithelioma in a male have been reported in the literature (Table I). This represents the twenty-fifth case which has been proved by autopsy and serial section of the testes, excluding those cases which showed evidence of testicular scars or cysts, and is the tenth case of mediastinal origin.

CASE REPORT

D. B., a 20-year-old white male college student, was admitted to The George Washington University Hospital on Sept. 4, 1956, because of a radiopaque shadow in the right hilar region found for the first time on routine x-ray two months prior to admission. The patient was asymptomatic except for occasional left posterior chest wall pain radiating anteriorly in the month prior to admission, and had no history of pulmonary disease, weight loss or anorexia.

The patient had had a right inguinal herniorrhaphy at one year of age, a left inguinal herniorrhaphy at age 6, and an appendectomy at age 7. The past history and review of systems were, otherwise, noncontributory. A chest roentgenogram taken in January, 1956, was negative.

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TABLE I

	AUTHOR	YEAR	AGE	SITE	GYNECOMASTIA	PREGNANCY TEST
1.	Askanazy[15]	1906	19	Hypophysis	--	--
2.	Miller and Browne[16]	1922	39	Retroperitoneal	--	--
3.	Arendt[17]	1931	20	Anterior mediastinum	Yes	--
4.	Kantrowitz[18]	1932	22	Mediastinum teratoma; chorioepithelioma; metastasis	No	Pos
5.	Fenster[19]	1934	27	Retroperitoneal	Yes	Pos
6.	Gerber[20]	1935	23	Retroperitoneal	No	Pos
7.	Weinberg[21]	1939	70	Bladder	No	Pos
8.	Mathieu and Robertson[22]	1939	27	Retroperitoneal	Yes	Not done
9.	Ferrarini[23]	1940	20	Retroperitoneal	No	Pos
10.	Erdmann, Brown, and Shaw[24]	1940	20	Retroperitoneal	No	Pos
11.	Hyman and Leiter[25]	1943	57	Bladder	--	Pos
12.	Plenge[26]	1944	30	Retroperitoneal	--	--
13.	Stowell, Sachs, and Russell[10]	1945	15	Diencephalon	No	Pos
14.	Laipply and Shipley[3]	1945	13	Mediastinal	Yes	Pos
15.	Hirsch, Robbins, and Houghton[8]	1946	26	Mediastinal	Yes	Pos
16.	Ferreira-Berrutti[27]	1946	37	Lung and liver; no definite primary	No	--
17.	Giroux and Desmeules[28]	1947	12	Mediastinal	No	Pos
18.	Dvoracek[29]	1949	24	Prostate	Yes	Pos
19.	Shlimovitz and Van Brown[5]	1950	27	Mediastinal	Yes	Pos
20.	Zorzi and Piacenti[30]	1952	29	Mediastinal	Yes	Pos
21.	Nielubszye[31]	1952	38	Mediastinal	Yes	Pos
22.	Lynch and Blewett[32]	1953	26	Mediastinal	Yes	*
23.	Pasquali[33]	1954	30	Retroperitoneal	Yes	Pos
24.	Lochmann[34]	1954	61	Liver	Yes	--

*Not mentioned in original case report.

There were no other children. His mother was living and well. His father was living six months post-hemicolectomy for adenocarcinoma. His mother had had a dermoid tumor removed from the right ovary soon after the patient's birth. The tumor was diagnosed at the time she was pregnant.

Physical examination revealed a well-developed, obese white male, weighing 240 pounds. Blood pressure 140/80 mm. Hg, pulse 74, respiration 20. There was hypertrophy of both breasts, but because of his weight this was difficult to classify as gynecomastia. A port-wine nevus was present over the right arm and shoulder. Healed inguinal herniorrhaphy and appendectomy incisions were present. The genitalia were normal. The physical examination was otherwise noncontributory.

Laboratory data: Hemoglobin 14.7, hematocrit 46, W.B.C. 9,100 with a normal differential; urine, normal; total blood proteins 7.5, A.G. ratio 1.08, albumin 3.92, globulin 3.6, Kunkel globulin 16.5; V.D.R.L. (serologic test for syphilis) negative; electrocardiogram normal.

X-ray showed an anterior superior mediastinal tumor. Skull plates and barium swallow were negative.

Hospital Course.—The patient had a right thoracotomy performed on Sept. 7, 1956, and a large globular irregular encapsulated mass was removed from the anterior superior mediastinum. The tumor was well encapsulated and there was no evidence of regional node involvement.

When the microscopic diagnosis was returned, showing areas of choriocarcinoma, a urine specimen was obtained for gonadotropin assay. On the fourth postoperative day this was quantitatively positive to 0.1 c.c. dilution. The patient's postoperative course was

complicated by pain in the left hip region. Roentgenograms suggested metastatic involvement of the left ilium, but no definite diagnosis could be made. He was discharged home on the eighth postoperative day.

Pathology Report.—The specimen was a single mass of tissue, lobular in shape, measuring 11.5 cm. by 9 cm. by 6.5 cm. The external surface was smooth, glistening and varied in color from light yellow to light red. An occasional irregular dark red hemorrhage was noted beneath the capsule. In some areas on the external surface, there was a suggestion of slight or gradual lobulation. The specimen had a soft, rubbery, consistency. The cut surface was soft, orange, and friable. It was focally necrotic and focally hemorrhagic. The periphery was more solid, had a yellow color and was nodular. The central softer necrotic areas appeared laminated and had the consistency of fibrinous material.

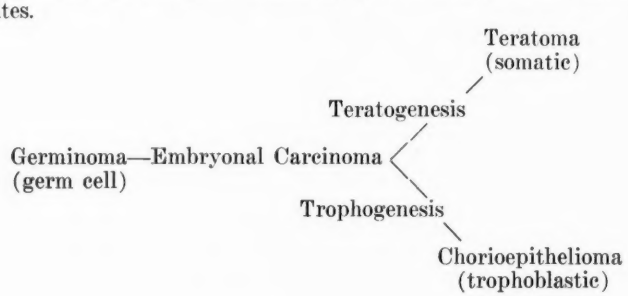
Microscopic diagnosis: Germinal tumor. The prime elements in the extragenital germinal tumor were seminoma and embryonal carcinoma, with areas of choriocarcinoma. The tumor appeared to have been completely removed, but vascular invasion within this tumor and the clinical course of similar cases made the prognosis uncertain. Thymic tissue was not identified.

The remainder of the patient's clinical course was rapidly downhill because of extensive skeletal and retroperitoneal metastasis. Deep x-ray therapy was administered at another institution, but he continued his downhill course, developing a cord compression paralysis which required a thoracic laminectomy from T_6 to T_8 on Oct. 9, 1957. The patient was re-admitted to The George Washington University Hospital on Jan. 18, 1957, in a comatose state, showing evidence of widespread metastasis, and he died Jan. 21, 1957.

Autopsy Findings.—Germinal tumor composed of seminoma, embryonal carcinoma, and chorioepithelioma, primary in the anterior mediastinum and surgically removed, with metastasis of the embryonal element to lungs, liver, bladder, esophagus, vertebrae, calvarium, left ilium, ribs, and brain. Bronchopneumonia, bilateral. Serial sectioning of both testes was negative for germinal tumor, but did show azospermia. Gynecomastia was not present.

DISCUSSION

The term extragenital chorioepithelioma is misleading. It is doubtful if any case actually represents a pure extragenital chorioepithelioma. Most cases represent chorioepitheliomatous elements in a germinal tumor, for the most part a teratocarcinoma. Recent studies have produced greater elucidation of the problem of teratogenesis. Most authors have conceded that gonadal teratoid growth originate from a totipotent germinal element of some sort, but extragenital teratomas have almost invariably been considered the result of aberrations in somatic development. How chorioepithelioma, which represents the trophoblastic or placental element, could be somatic in origin has always been difficult to assess. Recently, Friedman[11] has shown that extragenital germinal tumors are histologically identical to testicular tumors and, has, by comparative morphogenesis, set forth the following germinal origin and development, for both sites.



Most authors believe that extragenital chorioepithelioma arises from a multipotent or totipotent cell that has become separated at the morula or blastula stage, or from germinal cells in the urogenital ridge which in the 8 mm. embryo extends from the sixth cervical to the third lumbar region. The former could explain the bizarre extragenital locations reported, including the stomach, liver, and kidney. The latter explains the primarily retropleural or retroperitoneal location.

Since germinal extragenital tumors are by no means rare, it is surprising that so few with chorioepitheliomatous elements have been reported. This may be explained by the natural tendency for a short-life span of placental tissue.

Some authors, however, doubt if any case is truly extragenital.[12] They also site the length of the urogenital ridge and the fact that ectopic testicular tissue has been found in the adult along its extent, in addition to the normally located testicular tissue. The previously held theory of mediastinal germinal tumors being of thymic origin has also recently come under criticism. It has been shown that thymic carcinoma can microscopically strongly resemble germinal tumors, primarily seminomas and, in many instances, has been classified as such.[13]

If we accept the concept of development as diagrammed above, the finding of extragenital chorioepithelioma does not represent as dramatic a cellular aberration as previously considered. As most cases reported were in reality germinal tumors with various elements in addition to chorioepithelioma, it is questionable if such a case should ever be reported as an "extragenital chorioepithelioma." However, since such tumors do represent the trophoblastic phase of a germinal growth rather than the more common embryonal or somatic development, some justification for this terminology may still be present, particularly since so relatively few cases of trophoblastic response have been seen in male extragenital germinal tumors. Whether or not this is true will depend on how diligently chorioepithelioma is sought in other primary extragenital germinal tumors. If so, extragenital chorioepithelioma may be relatively common, and classifying it as a distinct entity unjustified.

The clinical response to a malignant germinal tumor will vary with the cellular predominance. If chorioepithelioma is appreciable, the hormonal and secondary sex changes are apparent and the survival time diminished. This element with its inherent invasive ability leads to widespread metastasis and rapid death. The elaboration of chorionic gonadotropin, which is specific to the chorionic villi is one of the characteristic diagnostic points. It produces the positive urine or serologic pregnancy test, and also gynecomastia, the latter having been experimentally reproduced in men, by Klatsen and Munson,[14] by daily administration of chorionic gonadotropin.

Most cases of extragenital chorioepithelioma develop varying degrees of testicular atrophy. These authors have also demonstrated the role of chorionic gonadotropin in the complex hormonal imbalance leading to this. Although frequently suggested as a means of determining postoperative residual tumor in resected cases, quantitative serial determinations of gonadotropin have not proved reliable as prognostic criteria. As illustrated in the above case, somatic

tumor elements may continue to progress despite a falling or negative quantitative gonadotropin determination. Since embryoma, seminoma, and teratoma have also been shown to give a positive urine or serum pregnancy test, this in reality may represent unidentified chorioepitheliomatous elements, since the responsible hormone is supposedly specific to the chorionic villi.

Radiologically, there is nothing characteristic of chorioepithelioma in the differential diagnosis of retroperitoneal or mediastinal tumors. However, if the possibility of chorioepithelioma is considered, the diagnosis can be made by a urine or serum pregnancy test in most cases before a tissue diagnosis is obtained. Although other germinal tumors do give a positive qualitative chorionic gonadotropin determination, chorioepithelioma gives a highly positive quantitative determination and in this manner can be differentiated from other germinal elements. Gynecomastia has been found to be present in over 50 per cent of the recorded cases. This may be unilateral in varying degrees of development, so that careful inspection is necessary. Although this tumor may occur at any age, as seen in the accompanying table, the greater number occur in the young adult. Since these tumors are from germinal anlage, and therefore dormant for many years, one may speculate that the hormonal effect of puberty gives some stimulus to the trophoblastic development of the germinal cells. It is interesting in this respect that this patient's mother developed a dermoid tumor of the ovary when pregnant with him, which was subsequently removed after his birth. An hereditary tendency for the development of germinal tumors in this family is suggested.

The treatment of chorioepithelioma is extremely unsatisfactory. Most patients die rapidly of metastasis. Lochmann[34] mentions several cases reported in the European literature of treatment with transfusions of blood from pregnant women. The theory is that even with a normal pregnancy, cells from chorionic villi invade the maternal vascular system stimulating an antibody response and a passive immune protection. Similar cases have not been reported in the American literature. Since chorioepithelioma is highly radioresistant, early diagnosis is imperative and radical excision the only treatment.

SUMMARY

This is believed to be the twenty-fifth reported case of primary extragenital chorioepithelioma in a male, proved by autopsy and serial section of both testicles. It is the tenth such case of mediastinal origin. We have reviewed the criteria for such a diagnosis and have listed the generally accepted cases.

The origin and morphogenesis of germinal tumors have been reviewed and while extragenital primaries have been in the past considered to be of somatic origin, recent workers have shown by comparative morphogenesis the germinal cell development of extragenital primaries as well as of genital primaries. We have pointed out that while most cases are reported as "extragenital chorioepithelioma," they are in reality germinal tumors in which chorioepitheliomatous elements have developed and it is questionable if these cases should be reported as a distinct entity.

The hormonal elaboration, age, and the development of gynecomastia have been briefly discussed and stressed as diagnostic criteria in these cases. Treatment consists of radical excision.

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MEDIASTINAL GRANULOMAS

A Revised Concept of Their Incidence and Etiology

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THIS paper, while dealing with a representative group of mediastinal tumors, is concerned not so much with the mere documentation of another series but more specifically with investigation of the true incidence and etiology of what is generally regarded as an insignificant segment of mediastinal neoplasia, namely, the granuloma.

Generally recognized is the fact that tumors of the mediastinum, although affording the roentgenologist a fascinating diagnostic exercise, almost invariably defy a precise preoperative diagnosis. Less widely appreciated, however, are the problems one may encounter in identifying and classifying any given tumor even with the specimen in hand and histologic sections analyzed. This is graphically demonstrated by the present series in which the gross pathologic findings were commonly deceptive and microscopic interpretation was too often nonspecific. Only after painstaking study has the etiology of some of these tumors been clarified.

As shown in Table I there have been thirty-seven operations for primary mediastinal tumors at this hospital in the past decade. On the whole this

Table I. Thirty-Seven Primary Mediastinal Tumors

TUMOR TYPE	BENIGN	MALIGNANT
Thyroid	2	–
Thymoma	2	2
Teratoma	–	3
Pericardial cyst	5	–
Lipoma	1	–
Neurogenic	4	2
Foregut cyst		
Bronchogenic	3	–
Esophageal	1	–
Granuloma	12	–
Totals	30	7

series is comprised of a fairly representative group of mediastinal tumors with the notable exception of an inordinately high number of granulomas. That this is an extraordinary reversal of the commonly accepted incidence of granulomas relative to other mediastinal tumors is best shown by comparing the

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incidence in our series (35 per cent) with the incidence reported in a recent comprehensive review[1] of mediastinal tumors in general (1.5 per cent). Considering that the latter figure was based on an analysis of 855 cases collected not from individual case reports but from all previously reported series, in other words an unselected group, one might conclude that the smaller figure provides a much more reliable estimate than does our own of the relative incidence of the mediastinal granuloma. This is supported by the rather meager literature on the subject, most of which has been devoted to the rarity and bacteriologic inscrutability of these tumors. Judging from our own series, even their granulomatous nature is liable to be missed. Although conceding that our experience is too limited to permit any emphatic conclusions, we suspect nonetheless that more careful study will establish both the true prevalence and, in many instances, the specific cause of the mediastinal granuloma. Such has proved true in our own investigations.

Grace[2] (1942) is credited with being the first to report the surgical excision of a so-called tuberculoma of the mediastinum. In 1944, the rarity of the lesion and its presumably tuberculous origin were further attested by a report by Blades and Dugan[3] of a posterior mediastinal tuberculoma in which only a very few acid-fast bacilli could be demonstrated. In 1950, Samson and coworkers[4] presented 4 more resected "tuberculomas" but raised some doubt about their exact etiology. Apparently the first ones to suggest that this might be a relatively common lesion were Storey and Lyons[5] who encountered 6 cases in a short period of time. Further doubt as to the cause of such granulomas was expressed by the Mayo Clinic group in 1954.[6] To the 16 cases culled from the literature they added another 16 cases in none of which were they able to establish a specific cause. For this reason they championed use of the term granuloma rather than tuberculoma when referring to such lesions. Subsequent reviews[7, 8] have re-emphasized the surgical aspect of these tumors especially when symptomatic or when a more sinister tumor cannot be ruled out.

As far as we can determine there was never any attempt to incriminate histoplasmosis as a cause until Samson did so in 1955,[9] and in that particular case his conclusion was based purely upon strongly positive histoplasmin and negative tuberculin skin tests. That same year Puckett[10] and, later, Schwarz[11] were able to demonstrate Histoplasma in resected mediastinal lymph nodes. Moreover, from analogous granulomatous lymphoid masses in the pulmonary hilus *H. capsulatum* has been cultured[12] and seen in specially stained tissue sections.[13, 14] More recently, Gillespie[15] has reported a case in which a mediastinal granuloma producing superior vena caval obstruction was attributed to histoplasmosis because of a positive complement-fixation test.

An analysis of our own series of mediastinal granulomas is presented in Table II. The patients were all young adults whose ages varied from 19 to 36 years. There were 7 men and 5 women; 10 white patients and 2 Negroes. Noteworthy features in this group include (1) a very low percentage of correct preoperative diagnoses, (2) uniformly negative bacteriologic yield in those cases in which the granulomatous contents were cultured, (3) the frequency with which the pathologist mistook the lesion for some totally different tumor such as a cystic teratoma or bronchogenic cyst, (4) the remarkably high incidence of

TABLE

PATIENT	AGE	SEX	RACE	SYMPTOMS	SKIN TESTS			LOCATION	PREOPER. DIAGNOSIS
					TBC	HISTO	COCCI		
No. 1 (N. G. M.)	36	F	W	Cough of 2 months' duration	Not recorded			Rt. paratracheal	Lymphoma; bronchogenic cyst
No. 2 (J. L.)	26	F	W	Mild superior vena cava syndrome	Not recorded			Rt. paratracheal	Lymphoma
No. 3 J. E. H.)	27	F	W	None	Not recorded			Rt. paratracheal	Lymphadenitis; lymphoma; cystic teratoma
No. 4 (H. S. W.)	23	M	W	Pain and burning over lower precordium	Neg	+++	Neg	Rt. posterior mediastinum adjacent to esophagus	Esophageal tumor
No. 5 (W. A. S.)	24	M	W	None	+	++	Neg	Rt. paratracheal	Cystic teratoma
No. 6 (G. N. H.)	22	M	N	None	Neg	Neg	Neg	Rt. paratracheal	Lymphoma; bronchogenic cyst
No. 7 (F. M. H.)	22	M	N	Hemoptysis	Neg	++++	Neg	Rt. paratracheal	Lymphoma
No. 8 (M. C. F.)	26	F	W	None	Neg	Neg	Neg	Left mid-mediastinum between aorta and pulmonary artery	Lymphoma
No. 9 (Z. D. L.)	31	M	W	None	+	Neg	Neg	Right mid-mediastinum extending around hilus	Lymphoma
No. 10 (A. C. T.)	23	M	W	None	Neg	++++	Neg	Rt. paratracheal	Mediastinal cyst
No. 11 (A. M. C.)	19	F	W	None	Neg	Not recorded		Rt. posterior mediastinum centered at level of carina	Teratoma
No. 12 (A. M. P.)	21	M	W	None	Neg	++++	Neg	Left mid-mediastinum level of carina	Lymphoma

II

GROSS APPEARANCE	SURGICAL PROCEDURE	SPECIAL FINDINGS	INITIAL PATHOLOGIC DIAGNOSIS	FINAL DIAGNOSIS
Thick-walled cyst 6 cm. in diameter filled with "sebaceous material" adherent to trachea, superior vena cava, and azygos vein	Resection	--	"Cholesteatoma"	Granuloma
Dense fibrous mass 6 × 4 cm. in diam. adherent to trachea superior vena cava and azygos vein	Biopsied	--	"Cicatrix"	Granuloma
Thick-walled cystic tumor 5 cm. in diam. adherent to trachea, superior vena cava and filled with thick white material	Resection	--	Bronchogenic cyst	Histoplasmic granuloma
Dense matted mass of fibrous tissue adherent to esophagus	Biopsied	--	Sclerosing mediastinitis	Granuloma (sclerosing mediastinitis)
Cystic tumor 5 cm. in diameter adherent to trachea; caseous content	Resection	--	Tuberculoma	Histoplasmic granuloma
Solid homogeneous mass 5 × 4 × 3 cm. with reddish-brown granular cut surface	Resection	Normal angiocardiogram	Infectious mononucleosis	Granuloma
Mass of matted lymph nodes measuring 8 cm. in diameter; middle lobe studded with subpleural nodules	Biopsied	--	Tuberculoma	Histoplasmic granuloma
5 × 3 × 3 cm. mass of lymph nodes	Resection	--	Boeck's sarcoid	Boeck's sarcoid
Large mass of matted lymph nodes	Biopsied	Refractile crystals seen with polarized light	Silicotic granuloma	Silicotic granuloma
Multiloculated thick-walled cystic mass 4 cm. in diam. with shaggy inner wall and thick pasty sebaceous contents	Resection	--	Cystic teratoma	Granuloma
10 × 5 cm. bilobulated cystic mass containing thick white creamy material; adjacent margin of rt. lower lobe presented a subpleural nodule	Resection	"Rare acid-fast bacillus"	Tuberculoma	Histoplasmic granuloma
4 cm. mass of lymph nodes with thick white creamy contents; adherent to vagus nerve	Resection	--	Tuberculoma	Histoplasmic granuloma

strongly positive histoplasmin reactors and, finally, (5) routine failure to establish a specific cause other than in that one case in which acid-fast bacilli were supposed to have been demonstrated. Prompted by previous experience in demonstrating fungi in various culturally-negative granulomatous foci,[16, 17] the tissue blocks from each of these cases were restudied in order to see what part histoplasmosis might play in their etiology.

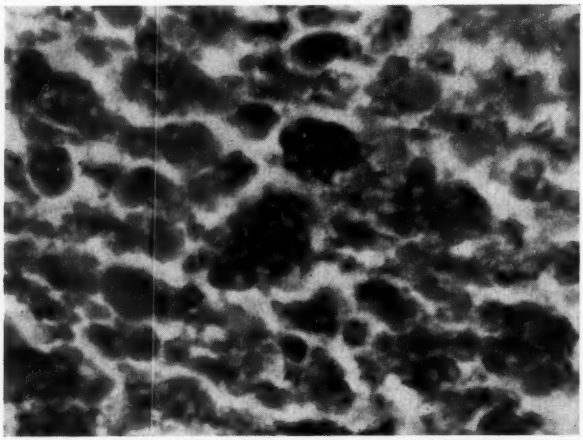


Fig. 1.—High-power photomicrograph of the necrotic center of the mediastinal granuloma resected in Case 4 (periodic acid-Schiff [PAS] stain). Innumerable typical Histoplasma are seen, some filling the cytoplasm of macrophages, others lying free in the surrounding debris. These organisms, none of which were identifiable in hematoxylin and eosin stained tissue sections, measure 3 to 5 microns in diameter and present a thin outer capsule. Many contain a small crescent-shaped mass of chromatin material usually situated eccentrically and staining intensely red by the PAS method.

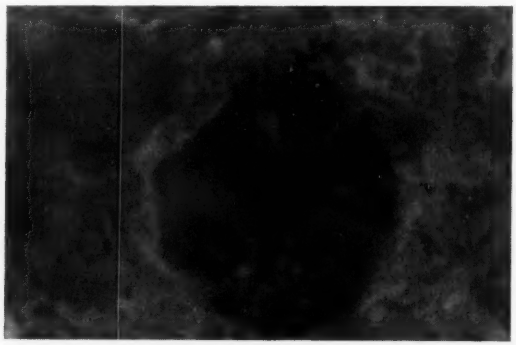


Fig. 2.—Photomicrograph showing a small cluster of Histoplasma lying within a degenerated macrophage. The faint outline of additional, less distinct, yeast forms can be seen outside the cell. In some instances a prolonged search may be required before finding diagnostic forms.

Utilizing the periodic acid-Schiff (PAS) stain it was possible to demonstrate Histoplasma in 4 cases. Organisms when seen were invariably found in areas of caseous necrosis (Figs. 1 and 2). In one granuloma they were present in

abundance while in the other 3 cases the visualization of diagnostic forms required persistent microscopic search. Despite careful study of the other 8 mediastinal granulomas no fungi could be found. Particular attention was devoted to the "tuberculoma" previously purported to contain acid-fast bacilli, for the tuberculin skin test had been negative in that case and we, in our reinvestigations, had been unable to corroborate the presence of acid-fast organisms. Therefore, as a final maneuver, tissue sections from this lesion were also restained by the methenamine-silver nitrate technique of Gomori.[18] A few scattered Histoplasma were then discernible. In addition, a small granulomatous focus excised from the lung immediately adjacent to this particular lesion was found to contain many Histoplasma (Fig. 3), thus indicating a primary complex type of histoplasmosis having both a parenchymal and a mediastinal component. Such an experience suggests that, whenever a similar mediastinal tumor is removed, any associated parenchymal focus should also be excised as a possible aid in diagnosis.

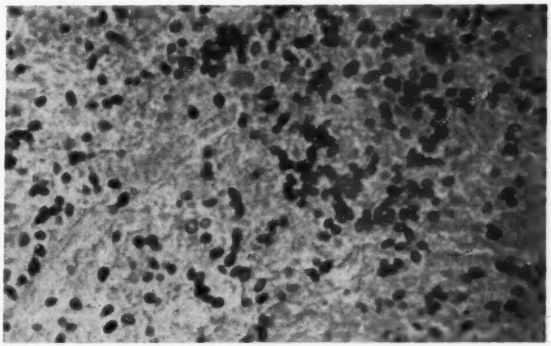


Fig. 3.—Photomicrograph showing numerous Histoplasma (Gomori's methenamine-silver nitrate stain). The capsule stains black with the chromatin mass often remaining inconspicuous. In our experience this has been the most satisfactory method for demonstrating organisms, being more sensitive, less capricious, and affording better contrast than the PAS and Gridley techniques. Another advantage is that the methenamine-silver nitrate method includes a decalcifying process, so that calcium granules which in other stains present confusing artifacts, resembling yeast forms of *H. capsulatum*, stain only a light brown or not at all.

Thus, of twelve mediastinal granulomas, five were due to histoplasmosis (Figs. 4-8). Two other cases were associated with positive skin tests to histoplasmin (Cases 4 and 10) but were histologically negative for fungi. Perhaps significant is the fact that in neither of these two specimens were there any zones of caseous necrosis, which in one instance (Case 10, Fig. 9) was due to evacuation of the tumor's necrotic contents in the process of excision and fixation of the specimen, while in the other (Case 4, Fig. 10) the pathologic process appeared "burnt out" and consisted mainly of fibrosis. There is the possibility, then, that these latter granulomas also represented residual histoplasmic foci, although in the final analysis we have grouped them among the nonspecific granulomas. Among the other granulomas, one (Case 6) presented a distinctive

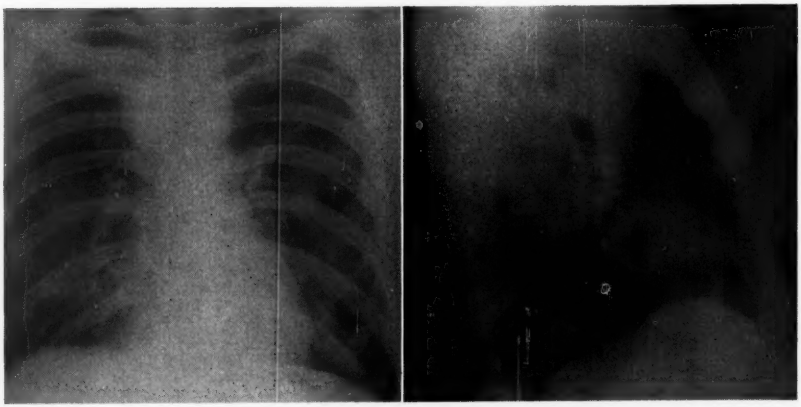


Fig. 4.—Case 5. A 24-year-old asymptomatic white man whose right paratracheal lesion was discovered in a routine chest x-ray. Both tuberculin and histoplasmin skin tests were slightly positive. A cystic granuloma was resected, the original pathologic diagnosis being "tuberculoma," the final one being histoplasmic granuloma.

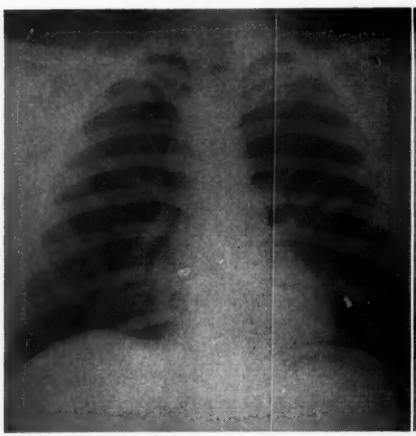


Fig. 5.

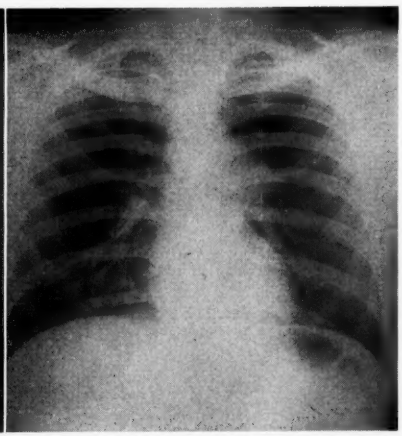
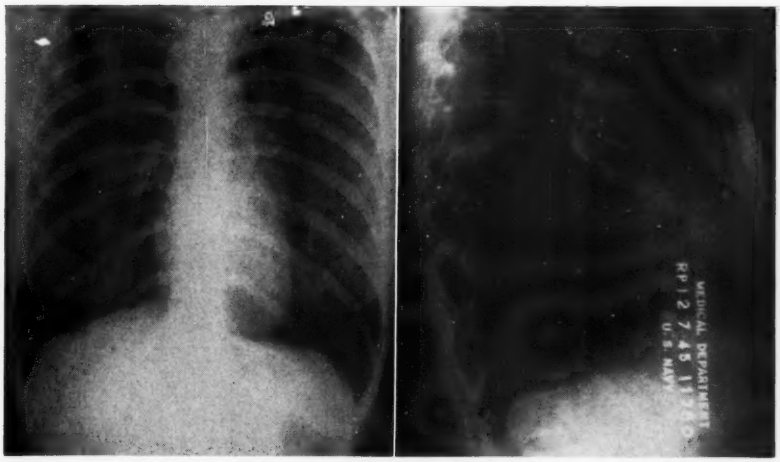


Fig. 6.

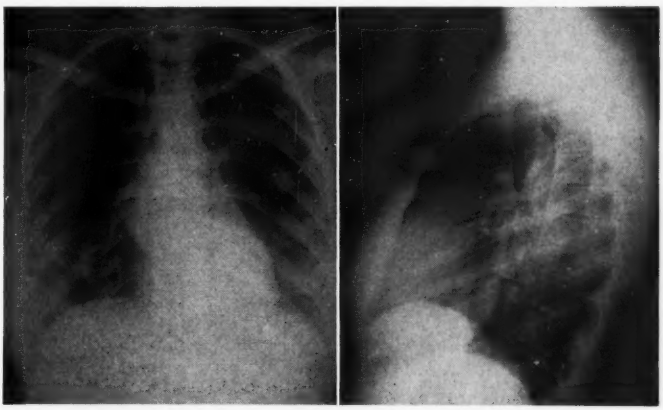
Fig. 5.—Case 7. A 22-year-old Negro admitted to the hospital because of recent hemoptysis. Chest roentgenogram disclosed a right paratracheal mass. All preliminary studies were negative except for a strongly positive histoplasmin skin test. At operation a granuloma was encountered, initially diagnosed as a "tuberculoma" but later found to contain Histoplasma.

Fig. 6.—Case 12. A 21-year-old asymptomatic white man who in a routine x-ray was found to have a left hilar mass. With the exception of a strongly positive histoplasmin skin test all other investigations were negative. This lesion was also considered a "tuberculoma" until subsequent study by special staining methods revealed typical Histoplasma.



A. *B.*

Fig. 7.—Case 3. A 27-year-old white woman without any symptoms whatever. Past history and physical examination were negative. At thoracotomy a cystic tumor filled with thick white material was dissected off the trachea and superior vena cava. This was misdiagnosed as a bronchogenic cyst. Histoplasma were demonstrated later. Note the small calcific parenchymal lesion seen in the lateral view just behind the sternum.



A. *B.*

Fig. 8.—Case 11. A 19-year-old asymptomatic white girl. Routine posteroanterior chest roentgenogram (*A*) showed a large round tumor mass projecting from the right hilus. Lateral x-ray (*B*) localized it to the mid-mediastinum beneath the carina. The tuberculin skin test was negative. The resected cyst contained thick creamy material and a few acid-fast bacilli were reported to have been found in its caseous contents. An adjacent subpleural granuloma was also excised from the right lower lobe. Meticulous restudy failed to reveal any Histoplasma by the PAS method, but neither could the presence of acid-fast bacilli be corroborated by Ziehl-Neelsen technique. Utilizing the methenamine-silver nitrate stain, a few forms diagnostic of Histoplasma were found in the mediastinal granuloma, while myriads of similar organisms were demonstrated in the associated pulmonary lesion (see Fig. 3).

type of reactive lymphoid hyperplasia (Fig. 11) which may cause the lesion to be mistaken for a thymoma, a danger emphasized by Castleman[19] and Iverson.[20] Another contained noncaseating granulomas typical of sarcoidosis (Fig. 12), while in another (Fig. 13) the polarizing microscope revealed a large number of doubly refractile particles, 1 to 3 micron in length, typical of silica crystals.

It seems quite logical to assume, therefore, that a certain percentage of mediastinal granulomas are the result of healed or healing mycotic infections. If the two can be considered comparable, one might infer that the mediastinal "tuberculoma" like the pulmonary "tuberculoma," is more commonly due to histoplasmosis and coccidioidomycosis than to tuberculosis. Thus, among 98

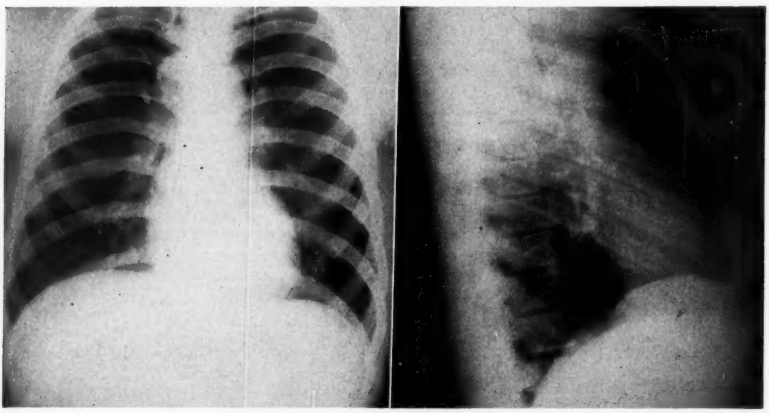
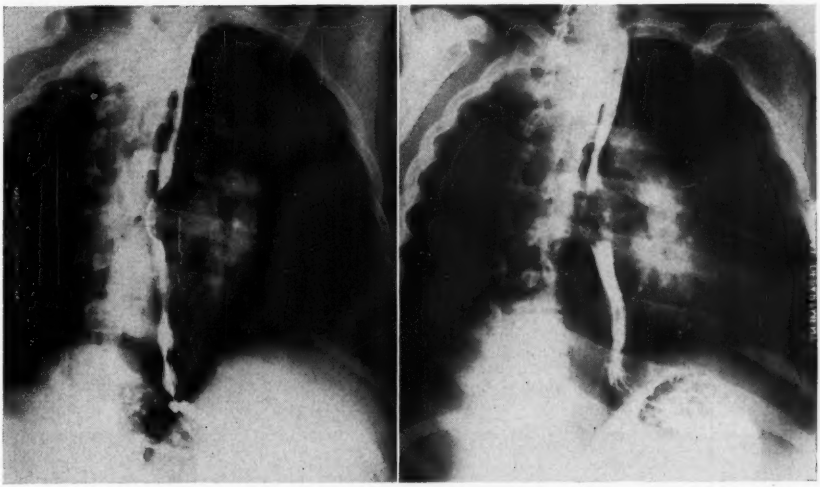
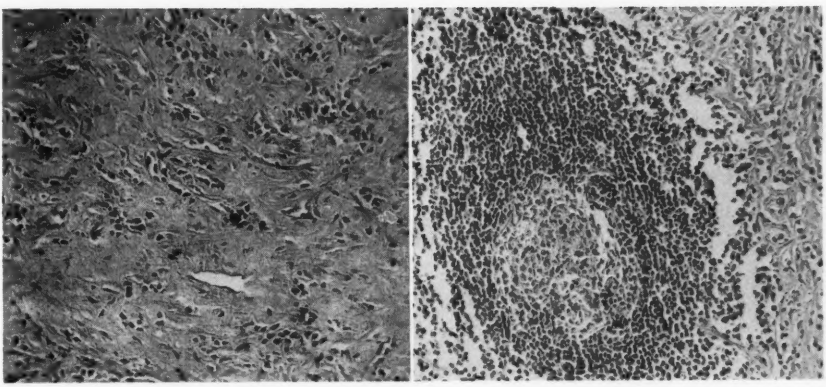


Fig. 9.—Case 10. A 23-year-old white man who had no symptoms. Histoplasmin skin test was strongly positive. The resected granuloma occupied a characteristic location alongside the trachea and superior vena cava to which it was adherent. Re-examination of the specimen showed that it was not a dermoid cyst as had been originally reported but a granuloma instead. Unfortunately its pasty contents had been evacuated, thus obviating a specific diagnosis.

pulmonary granulomas analyzed by one of us,[21] Histoplasma were found in 53 (54 per cent), Coccidioides in 5 (5 per cent), and tubercle bacilli in 15 (15 per cent). The fact that cultures were consistently negative for fungi is probably a reflection of the "burnt out" character of these lesions. This is supported by the failure of the organisms to stain by routine methods, which in itself is a sign of nonviability. On the other hand, it may be necessary to use special cultural techniques similar to those used by Emmons and his co-workers,[22, 23] who obtained cultural proof that in at least one endemic zone 50 per cent of apparently normal dogs show histoplasmic involvement of mediastinal lymph nodes. Obviously, it is a common infection. The question now arises as to how frequently may such fungal involvement resolve so completely as to leave no radiographic and minimal anatomic evidence of previous histoplasmic lymphadenitis.



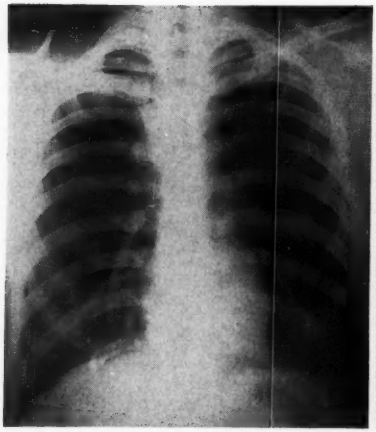
A. *B.*

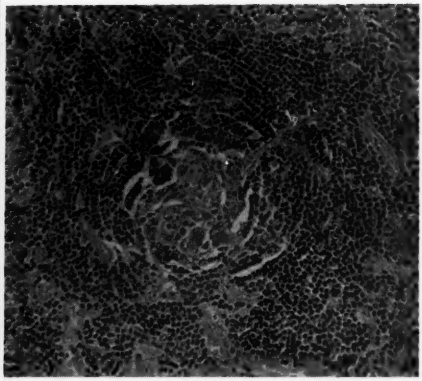


C. *D.*

Fig. 10.—Case 4. A 23-year-old white man whose only complaint was a burning sensation over the lower precordium. Examination was negative. Tuberculin skin test was negative; histoplasmin was strongly positive. Barium swallow demonstrated a smooth compression of the mid-esophagus thought to be consistent with a benign intramural esophageal tumor (*A* and *B*). At operation a firm mass of fibrous tissue was encountered. Microscopic examination revealed a large amount of dense collagenous connective tissue (*C*). Scattered throughout were small focal collections of chronic inflammatory cells and carbon particles. (*D*) Lymphoid follicles are seen presenting a prominent germinal center surrounded by a cuff of mature lymphocytes, the follicles being surrounded by a mass of connective tissue. This picture is characteristic of sclerosing mediastinitis (R. Shuman, M.D., Armed Forces Institute of Pathology). No fungi were seen with special stains.

A.

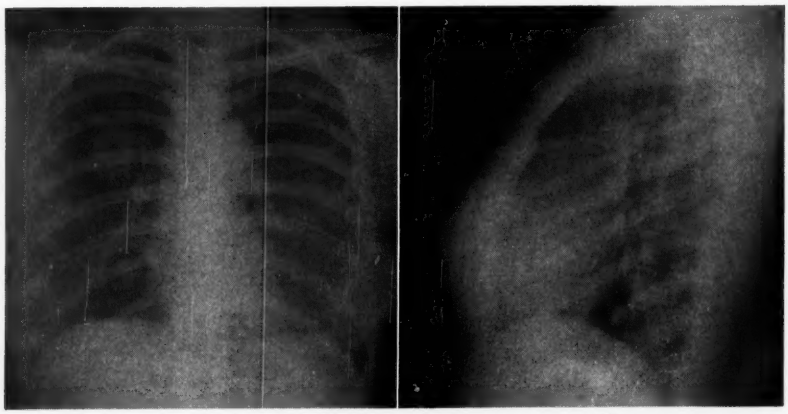




B.

Fig. 11, *A* and *B*. *(For legend see opposite page.)*

A.



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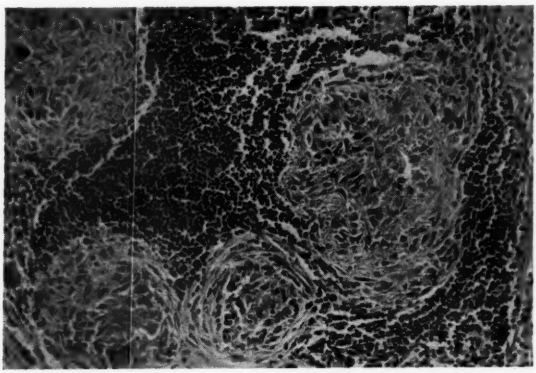
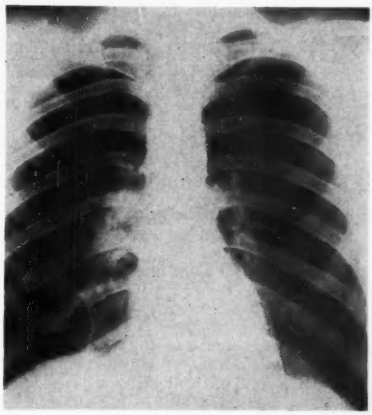


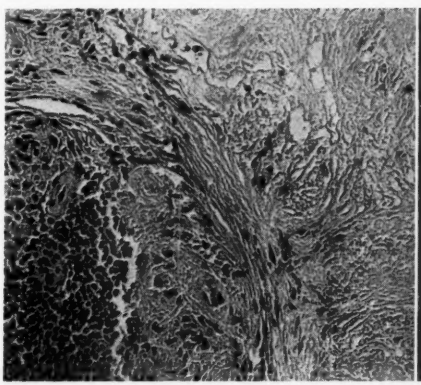
Fig. 12, *A* and *B*. *(For legend see opposite page.)*

SUMMARY

Of 37 primary mediastinal tumors in patients operated upon at the U. S. Naval Hospital, Bethesda, Md., 12 were granulomas, a relative incidence far higher than that generally ascribed to this lesion. Two of these had been previously misdiagnosed, one having been called a cystic teratoma and the other



A.



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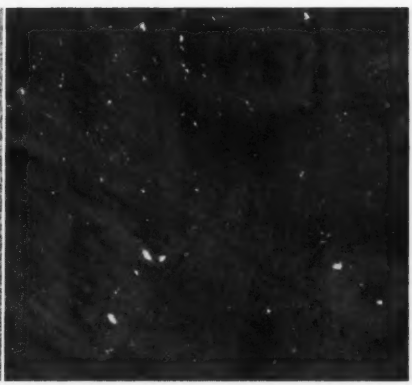

C.

Fig. 13.—Case 9. A 31-year-old white man who presented with an asymptomatic mass in the right hilus (*A*). A large mass of matted lymph nodes were encountered at thoracotomy. Microscopically, there were prominent groups of lymphocytes surrounded by epithelioid cells. These are separated by a layer of fibroblasts from areas of fibrillary eosinophilic material in which there are a few blood vessels, macrophages, and a large amount of carbon pigment (*B*). With polarized light the central pigmented areas are found to contain many small (1 to 3 microns) doubly refractile particles resembling silica (*C*).

Fig. 11.—Case 6. A 22-year-old Negro without any symptoms related to right paratracheal mass (*A*). The resected specimen presented many lymphoid follicles with germinal centers containing large acidophilic masses as well as the usual large reticulum cells (*B*). With differential staining, these masses are identified as collagen. Cuffs of small mature lymphocytes surround the germinal centers. The endothelial lining cells of the sinusoids are swollen, giving the appearance of small groups of epithelial cells with a lymphoid stroma. Superficially this lesion has some resemblance to a thymoma.

Fig. 12.—Case 8. A 26-year-od asymptomatic white woman whose chest x-ray showed a left hilar mass (*A*). Microscopic examination of the resected specimen disclosed many granulomas varying in size and presenting a thin laminated layer of fibroblasts at the periphery surrounded by chronic inflammatory cells (*B*). Epithelioid cells and giant cells are noted, but no areas of caseous necrosis are seen. Since no organisms could be demonstrated, this lesion falls into that group of granulomatous reactions called Boeck's sarcoid.

a bronchogenic cyst. Most of the others were presumed to be tuberculomas, although cultures had been uniformly negative and acid-fast bacilli were demonstrated in only one.

Re-examination of these granulomas by the periodic acid-Schiff stain has demonstrated Histoplasma in 4 cases. In still another case, Histoplasma were found only after resorting to Gomori's methenamine-silver nitrate stain. Two other lesions associated with strongly positive histoplasmin and negative tuberculin skin tests were negative for fungi as were the other 5 mediastinal granulomas.

It is suggested that histoplasmosis is more commonly the cause of mediastinal granulomas than has previously been thought.

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TUBERCULOMA OF THE MEDIASTINUM

Report of the First Culturally Proved Case

J. Winthrop Peabody, Jr., M.D., Washington, D. C., Harry E. Walkup, M. D., Oteen, N. C., and James D. Murphy, M.D., Baltimore, Md.

INCREDIBLE though it seems, a review of the literature on resected mediastinal tuberculomas fails to reveal a single instance in which tubercle bacilli could be cultured from the specimen.[1] Furthermore, in only one case were acid-fast bacilli demonstrated histologically.[2] For this reason Samson and his co-workers[3] and later Kunkel, Clagett, and McDonald[4] expressed doubt that these lesions were necessarily due to tuberculosis and preferred to call them granulomas in deference to their unknown etiology. Recent studies show that histoplasmosis accounts for a certain number of mediastinal granulomas and it has been suggested that the mediastinal granuloma like the pulmonary granuloma is much more commonly due to fungi than to *M. tuberculosis.*[1] In the following case which appears to be unique, the resected specimen was teeming with tubercle bacilli and cultures were readily positive for *M. tuberculosis.*

CASE REPORT

R. B. C., a 53-year-old white veteran, was admitted to the Veterans Administration Hospital, Oteen, N. C., for excision of a mediastinal tumor discovered during his recent hospitalization at VA Hospital, Mountain Home, Tenn., where he had been treated for a peptic ulcer.

The patient denied any respiratory symptoms other than a slight unproductive cough of approximately 5 years' duration. Past history and system review were not significant. Physical examination was not remarkable. Tuberculin (PPD No. 1) and histoplasmin skin tests were both positive.

Multiple roentgenograms of the chest revealed a large round partly calcified mass in the mid-mediastinum beneath the carina. The density measured about 8 cm. in diameter and although obscured in the posteroanterior projection by the cardiac silhouette, it was readily visualized in the lateral and oblique views as well as in tomograms (Fig. 1). There was widening of the carina with pressure on the right stem bronchus. A small calcific density was noted in the anterior basal segment of the right lower lobe. The roentgenologist's impression was a teratoid tumor or a mass of calcified tuberculous lymph nodes.

Operation.—With the patient in the left lateral decubitus position under endotracheal cyclopropane-oxygen anesthesia, the right pleural space was entered through the bed of the subperiosteally excised sixth rib. After dividing the inferior pulmonary ligament and retracting the lung upward, a firm tumor could be palpated beneath the carina anterior to the esophagus. The right vagus nerve was adherent to the mass and was divided. By a process of sharp and blunt dissection the tumor was freed from the right inferior pulmonary vein, right stem bronchus, and then from the carina and left stem bronchus, to all of which structures it was adherent. The entire tumor was removed, and the chest closed. Postoperative course was uneventful and the patient remains well after a course of antituberculous chemotherapy.

From the Surgical Service, Veterans Administration Hospital, Oteen, N. C.
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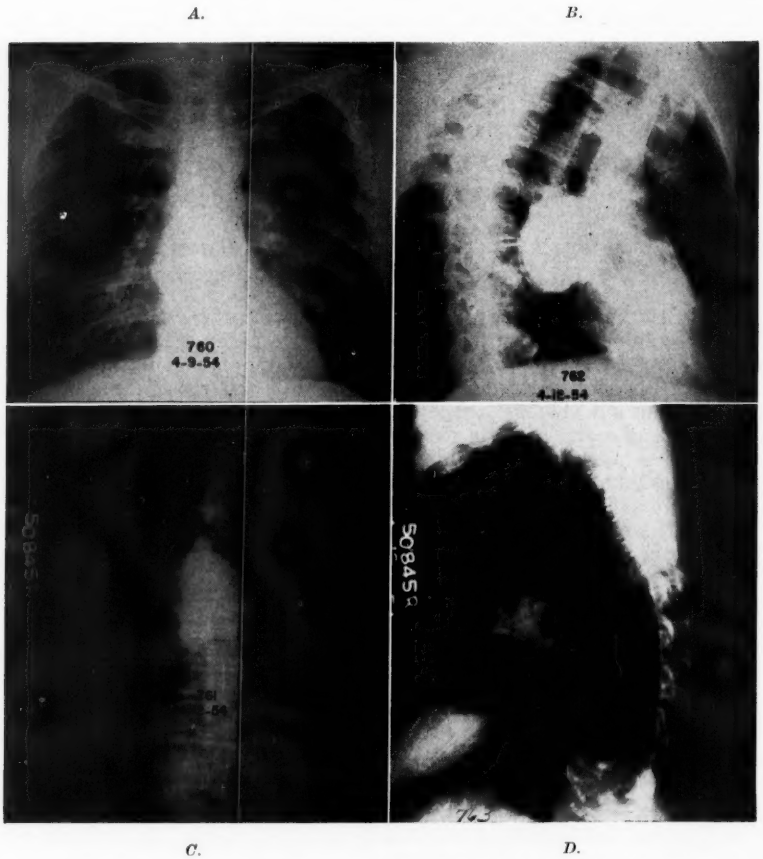

Fig. 1.—Chest roentgenograms showing the round partly calcified mid-mediastinal mass. Only the right margin of the tumor is seen in the posteroanterior projection (*A*) but oblique (*B*) as well as overpenetrated posteroanterior (*C*) and lateral (*D*) views show it quite clearly.

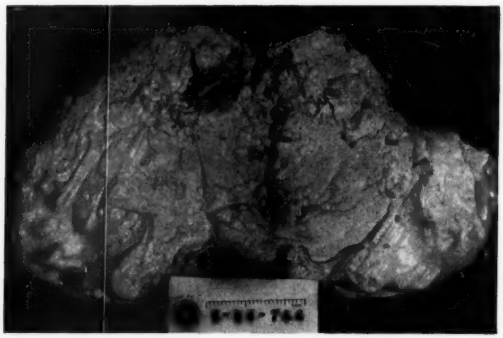

Fig. 2.—Photograph of the resected specimen. Cut surface reveals the "toothpasty" contents which were teeming with acid-fast bacilli on smear and from which *Mycobacterium tuberculosis* was readily recovered by culture.

Pathology.—Grossly, the tumor was a smooth, well-encapsulated mass measuring 6 cm. in diameter and weighing 190 Gm. It felt somewhat mushy except for one surface which presented a firm calcified exterior. On cut section the tumor was found to be filled with white caseous material resembling tooth paste (Fig. 2). The wall of the cyst was quite thin measuring 1 mm. in thickness.

Microscopically, the capsule of the cystic tumor was comprised of hyalinized fibrous tissue containing many islands of calcification some of which actually represented bone and contained marrow spaces. Scattered throughout the wall were small collections of lymphocytes. In one area there was distinct lymphoid structure containing anthracotic pigment. There was no epithelial lining.

Direct smears of the caseous contents reveal a great number of acid-fast bacilli. Cultures were positive for *M. tuberculosis.*

SUMMARY

A mediastinal tuberculoma is reported in which there was ample histologic and bacteriologic proof of its tuberculous origin. As far as the authors can determine this is the first such case to be reported.

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A SIMPLE METHOD OF SCAPULAR TRACTION FOR CLOSURE OF LATERAL OR POSTEROLATERAL CHEST INCISIONS

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OCCASIONALLY there is developed a simple, easy, but different method of doing some common, everyday process or task, which is worthy of reporting. In closing a lateral or posterolateral thoracic wound, the task of holding the shoulder in place is a difficult one for the assistant or for the anesthetist. If the shoulder is not held in proper position the muscles and skin do not lie properly for accurate reapproximation. It has been found by repeated practice that once the ribs have been approximated the inferior angle of the scapula, covered by its normal muscles, can be brought easily into view. An ordinary towel clip, clamped through the muscles into the bone of the inferior angle of the scapula, provides secure control of the shoulder (Fig. 1). With one hand it is easy for the assistant to keep the shoulder in proper position by traction on the towel clamp, leaving the other hand free for other tasks. The incision is unobscured, making approximation by suture simple and easy. Removal of the towel clamp is at times slightly awkward and therefore the muscles must not be sutured too closely to the towel clamp before it is removed.

SUMMARY

In the closure of a lateral or posterolateral thoracic incision a towel clamp applied to the inferior angle of the scapula easily provides the necessary traction for holding the shoulder in the proper position while the tissues of the wound are being accurately reapproximated by suture.

From the Department of Surgery, Church Home and Hospital, Baltimore, Md.
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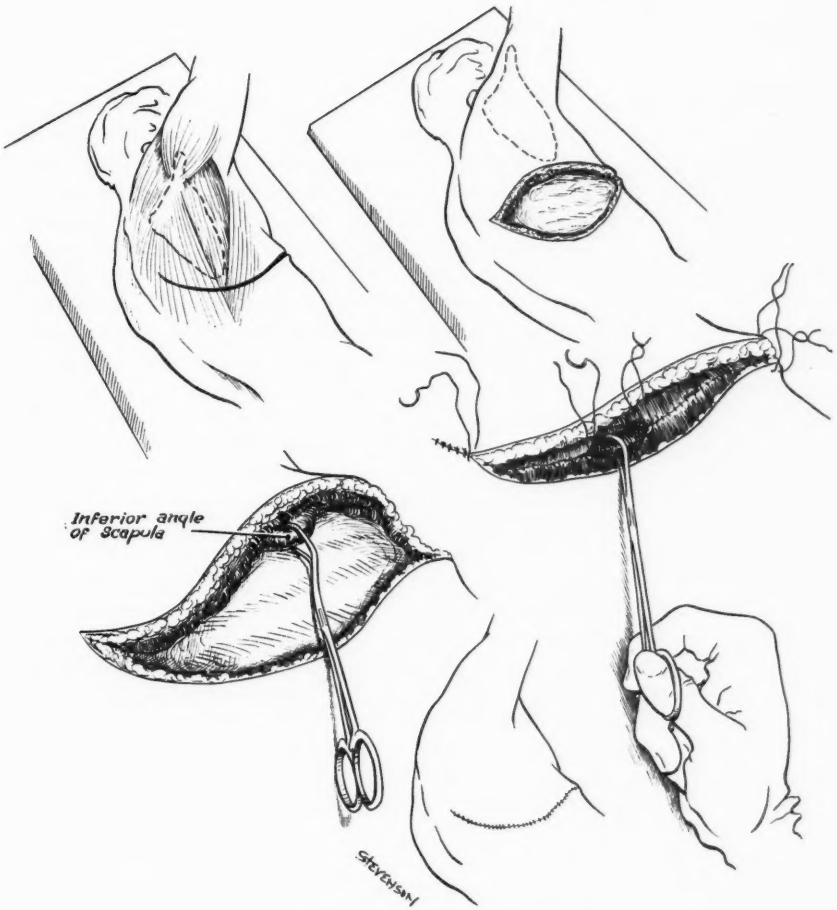

Fig. 1.—See text for explanation.

RESECTION IN PULMONARY CRYPTOCOCCOSIS (TORULOSIS)

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WALLA WALLA, WASH.

CRYPTOCOCCOSIS (torulosis, European blastomycosis, Busse-Buschke's disease, 1894) is a subacute or chronic, very often fatal, infection caused by *Cryptococcus neoformans*, a neurotrophic, yeastlike, nonsporulating, nonmycelial, budding fungus, 5 to 15 micra in diameter, characterized by a wide gelatinous capsule in India ink stain.[1-6, 31] The organism is of world-wide distribution, affecting both sexes and all ages.[43, 46] It should be differentiated from *Blastomyces dermatitides*, the cause of Gilchrist's disease, isolated in 1898,[1, 6, 7, 39, 40] and *Histoplasma capsulatum*, originally described by Darling in 1906.[49, 50] The infection is either localized or systemic. The organisms show a great predilection for the central nervous system, with manifestations of a subacute or chronic meningitis which is still almost invariably fatal.[1, 7, 8] The disease is not transmitted from man to man, from animal to animal, or from animal to man. The infection is believed to be air-borne from a reservoir in the soil.[31] The most common portals of entry appear to be the respiratory tract and the skin. Dissemination from a localized focus in the lung, skin, or other organs takes place through the blood stream and lymphatics. Small lesions in the lungs may heal by fibrosis and eventual calcification.[1, 5, 9, 32]

Primary cryptococcosis is not too uncommon, with more than 310 cases now on record in world's medical literature, and nearly 38 fatal cases a year recorded by the National Office of Vital Statistics during recent years.[8, 9, 25, 26, 28-31, 38] At least 100 reported cases had clinical manifestations of a pulmonary disease sometime during their courses, and approximately one third of these showed localization to one lobe or lung.[8, 9, 27] Since Dormer and his colleagues,[10, 48, 51] in 1945, first reported a case of localized pulmonary cryptococcosis treated by resection, there have been at least 25 additional cases, including our own, bringing the total to 26 cases reported, treated in this manner.[9, 11-15, 23, 24, 33, 35, 37, 41, 42, 48]

DIAGNOSIS

The diagnosis of pulmonary cryptococcosis of localized form seldom has been made before operation. There is reason to believe that the diagnosis has been missed not infrequently after operation and even after autopsy. A more routine use of special histologic staining techniques in every case of obscure pulmonary disease, particularly "coin lesion," "chronic granuloma," and those considered to be "resolving pneumonia" or "non-specific pneumonitis," will most likely result in the diagnosis of more cases.[11-13, 31]

From the Veterans Administration Hospital, Walla Walla, Wash.
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Clinically, pulmonary cryptococcosis most often simulates a low-grade subacute or chronic pneumonitis, tuberculosis, neoplasm, or lung abscess.[1, 5, 8] Chest roentgen findings usually fall under one of the following categories as described by Bonmati and his co-workers.[9]

1. Large, solitary rounded mass or several nodular lesions which may excavate. This includes "coin lesions," "toruloma," or pseudo-tumors.

2. Disseminated nodular lesions of miliary or slightly larger size, frequently with linear infiltrates.

3. Single or multiple infiltrates which may be either patchy or linear in character.

There is no dependable skin test. Diagnosis may be proved by identifying the organisms in the sputum, pleural fluid, or bronchial aspirations, by their isolation in cultures grown in Sabouraud's medium at 37° C., and establishing their pathogenicity by inoculation into mice or guinea pigs.[1, 12] The use of special tissue staining techniques, such as the periodic acid-Schiff, Gomori methenamine-silver preparation, and the mucicarmine stain, is essential.[11, 16, 31] Ordinary hematoxylin and eosin sections may show the organisms as poorly defined, yeastlike structures with occasional budding, located mostly intracellularly in macrophages or giant cells, or extracellularly in necrotic centers of the granulomas. The special stains mentioned give sharper definition so that the organisms are more readily identified. In the experience of the Armed Forces Institute of Pathology, with the exception of occasional Blastomyces, the cryptococcus is the only one of the more commonly encountered fungi that takes the mucicarmine stain.[16, 31]

INDICATIONS

In the absence of central nervous system involvement, we believe that all patients with localized pulmonary cryptococcosis are definite candidates for excision of their pulmonary lesions, unless they are suffering from other hopeless systemic disease, or are not acceptable surgical risks. Localized pulmonary disease is interpreted to mean that the lesion or lesions do not involve more than one lobe of either lung, or not more than one lung, if the lesion is multilobar. The more localized the lesion in the lung, the greater the indications for excision. Although less ideal, the indications may be extended to include exceptional cases with very well-localized bilateral nodular or "coin" lesions which do not involve more than one lobe of both lungs, so that bilateral pulmonary resection is still possible. Upon fulfillment of the above criteria, pulmonary resection should not be delayed. Lobectomy is the procedure of choice if the lesion is unilobar. Wedge or segmental resection is less desirable because of the greater likelihood of incomplete removal of a lesion caused by organisms which have a great tendency to invade the central nervous system. Pneumonectomy is indicated in the presence of multilobar unilateral disease in which all lobes of one lung are involved.

Should adequate preliminary diagnostic studies fail to reveal the nature of a localized pulmonary lesion which may be of cryptococcal origin, the lesion

should then be considered an indeterminate one. Unless there are overwhelming contraindications such as those already alluded to, the presence of an indeterminate pulmonary lesion is an indication for an exploratory thoracotomy and excision.

Since at present there is no entirely satisfactory medical treatment for the almost invariably lethal cryptococcal meningitis, in spite of iodides, actidione, stilbamidine, modern chemotherapeutic agents and antibiotics.[5, 7, 15, 17-20, 31] excision of a localized pulmonary lesion must be done early, before the development of meningitis or central nervous system disease. Approximately 75 per cent of the patients in 100 reported cases of pulmonary cryptococcosis eventually died with central nervous system involvement.[9, 10, 28] Resection for localized pulmonary cryptococcosis should be considered in the same sense as resection for bronchogenic carcinoma, as proposed by Taber,[21] Froio and Bailey,[14] Berk and Gerstle,[13] and Poppe.[33, 47] In Australia, where cryptococcosis is better recognized, pulmonary lesions are resected, not only as a prophylaxis against nervous system involvement, but also in the hope that resection will affect the course of the disease more favorably even when meningitis is already present. Clinical improvement and apparent recovery have been reported following resection in the presence of cryptococcal meningitis in at least 2 cases by McConchie[37] and Susman.[36, 52] The follow-up periods have been 6 months and about 5 years, respectively. Susman's patient received postoperative roentgen therapy to the skull and entire spine, with a total skin dose of 6,900 r, given expectantly.[52, 53] Cryptococcal meningitis or meningoencephalitis is known for its chronicity and long periods of remission.[44, 45]

CASE REPORT

A 37-year-old white male Army veteran was admitted to the Veterans Administration Hospital, Walla Walla, Wash., on Dec. 16, 1954, with complaint of right chest pain for the previous 3 months. A survey roentgenogram of the chest with a portable unit had revealed the presence of a lesion in the right lung approximately 3 months before the patient came to the hospital. The patient had received a course of antibiotics just prior to admission, prescribed by a local physician for treatment of what was believed to be a right pneumonia. Low-grade fever had been present intermittently since the onset of the right chest pain. The patient had smoked one and a half packages of cigarettes daily for many years. The only positive physical findings on admission consisted of some restriction of inspiratory expansion of the right side of the chest and the presence of harsh breath sounds over the right upper half of the chest. Râles were absent.

Laboratory data on entry showed a hemoglobin of 13.2 Gm., white blood cell count of 13,800, with 72 per cent neutrophils. Blood sedimentation rate was 21 mm. The intradermal tuberculin PPD first and intermediate strengths, coccidioidin, and histoplasmin tests were negative. PPD second strength was weakly positive. Repeated sputum examinations for acid-fast organisms on smear and culture were negative.

A miniature survey film of Sept. 24, 1954, revealed a small area of confluent infiltration at the third anterior interspace on the right, and a subsequent film of Oct. 19, 1954, showed no essential change. A film of Dec. 6, 1954, disclosed a small central highlight within the area of infiltration and, at the same time, marked enlargement of the right hilus. On Dec. 13, 1954, there was further increase in the right parahilar density, which now

closely approximated the lesion originally seen. On Dec. 22, 1954, there was further enlargement and apparently complete coalescence of these two lesions, thereby forming a large area of uniform haziness at the mesial aspect of the right mid-lung field. On lateral planigrams the lesion appeared to be wedge-shaped, mainly involving the superior segment of the right lower lobe; however, the posterior and lateral basal segments appeared to be involved also. By Jan. 3, 1955 (Fig. 1), there was a definite decrease in the size of the area of uniform density, especially at its parahilar aspect.

Bronchoscopy, on Dec. 20, 1954, was negative except for a moderate amount of white sputum from the right lower lobe. A right anterior scalene lymph node biopsy was negative. It was felt that bronchogenic carcinoma could not be ruled out.

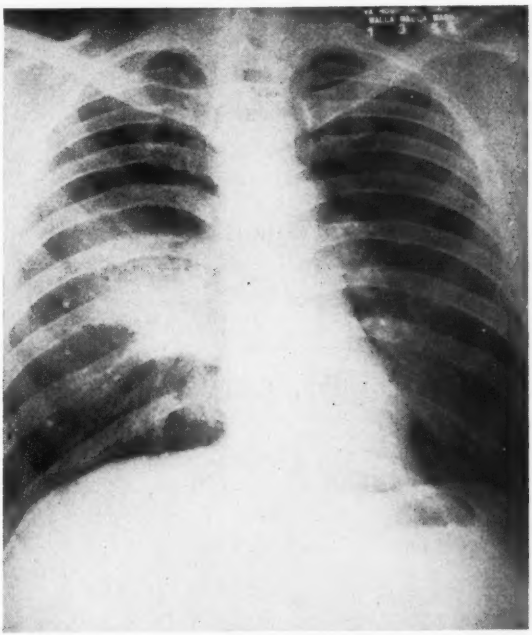

Fig. 1.—Preoperative chest roentgenogram showing right lower lobe lesion, mostly in superior segment.

On Jan. 14, 1955, a right exploratory thoracotomy was performed, combined with biopsy and frozen section studies from two nodular lesions within the right lower lobe, followed by a right lower lobectomy. The report on frozen sections was that of a granulomatous or inflammatory lesion.

Gross examination of the excised right lower lobe revealed a firm, atelectatic superior segment which contained multiple abscesses filled with a thick yellow liquid. There were other patchy areas of consolidation within the medial and posterior basal segments, and at least two other small, hard, calcified nodules in the remainder of the basal segments. Microscopic report by the Armed Forces Institute of Pathology, Washington, D. C., indicated a granulomatous lesion (Figs. 2 and 3) containing many yeastlike organisms with occasional budding, mostly within giant cells and macrophages. The organisms were well defined by the Gomori methenamine-silver preparation. Mucicarmine stain demonstrated the organisms to be carminophilic. The diagnosis was pulmonary cryptococcosis.

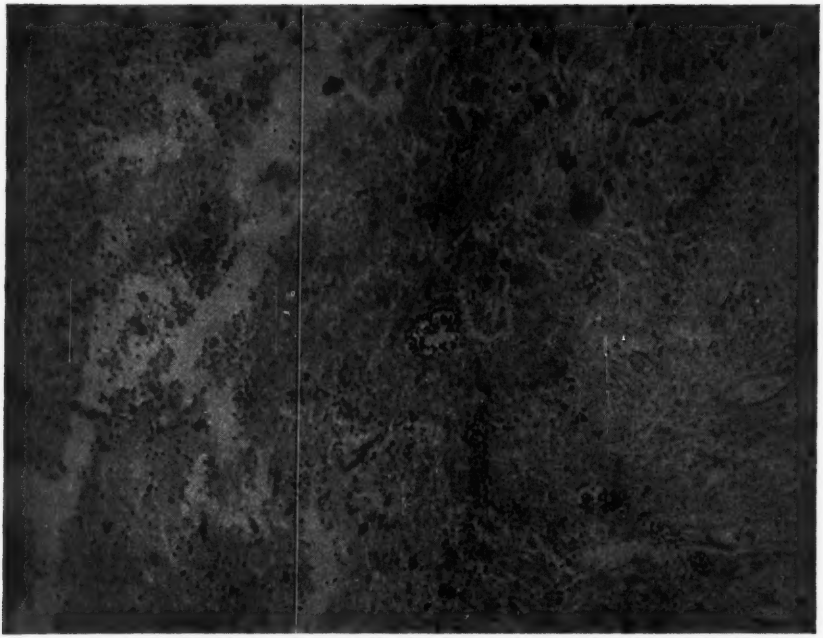

Fig. 2.—Low-power microscopic view of granulomatous appearance of lesion with giant cells. (Hematoxylin and eosin stain ×100; reduced ⅙.)

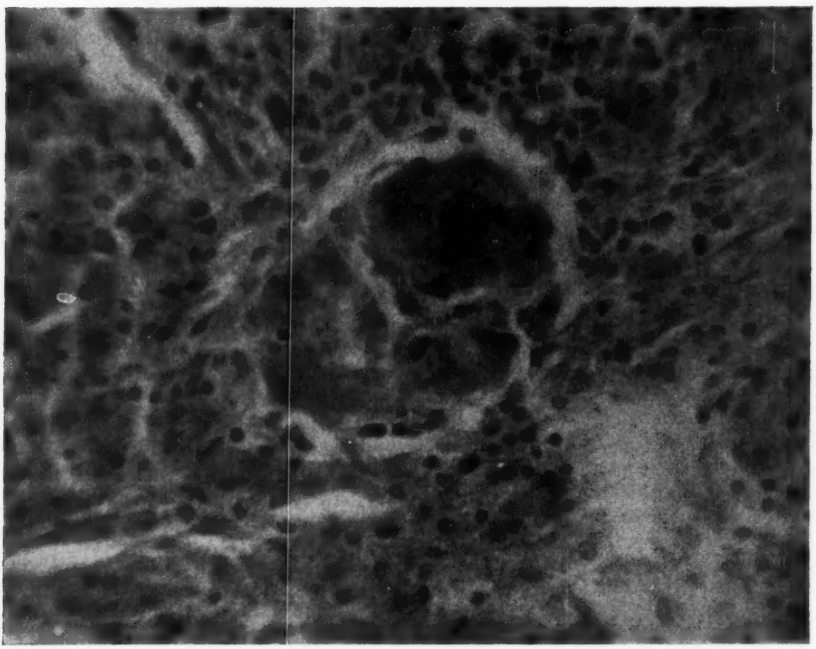

Fig. 3.—High-power microscopic view of giant cell laden with white, yeastlike *Cryptococcus neoformans*. (Hematoxylin and eosin stain ×430; reduced ⅙.)

The postoperative course was entirely uneventful. The patient was sent home on the twelfth postoperative day. He soon returned to work and has remained entirely well. Follow-up films of the chest in the second postoperative week and 17 months after lobectomy (Fig. 4) revealed no recurrence. The patient remains entirely well 28 months after the operation, and is now working in Anchorage, Alaska.

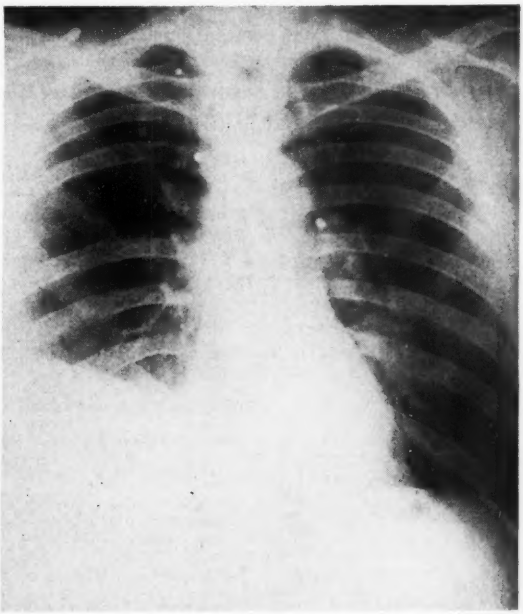

Fig. 4.—Roentgenogram of chest 17 months after right lower lobectomy.

RESULTS AND DISCUSSION

Table I shows the reported cases of resection for localized pulmonary cryptococcosis in available literature.

Significant is the fact that 19 of the 21 reported survivors or apparent cures had no central nervous system manifestations prior to surgical excision of localized pulmonary lesions.[9-14, 33, 35-37, 41, 42] The importance of long-term follow-up of resected cases before cures can be claimed was well illustrated by the case reported by Palmrose and Losli.[23] Their patient developed cryptococcal meningitis more than 3 years after enucleation of a right lower lobe lesion and died during the following year. One can only conjecture whether or not the course of the disease would have been altered more favorably if a lobectomy, instead of a simple enucleation, had been performed.

SUMMARY AND CONCLUSION

1. Of the 26 reported cases of sufficiently localized pulmonary cryptococcosis treated by resection, 21 (81 per cent) survived free of symptoms and

TABLE I

AUTHOR AND YEAR REPORTED	CASES	APPARENTLY CURED SURVIVORS	RESULTS
Dormer et al., 1945 and 1947	2	1	One alive and apparently well 3 months later, in spite of postoperative c.n.s. disease which responded to large doses of iodides; other died with c.n.s. disease 31 days later, in spite of iodides
Starr and Geddes, 1949	1	1	Alive and well 7 years later
Froio and Bailey, 1949	1	1	Alive and well more than 13 years later
McConchie, 1951	3	3	One asymptomatic for 10 years with undiagnosed small circumscribed lesion in contralateral lung; other with c.n.s. disease before resection improved and apparently well 6 months later; another alive and well 2 years later
Palmrose and Losli, 1952	1	0	Died with c.n.s. disease 4 years later, with onset of symptoms more than 3 years after enucleation of lesion
Cruickshank and Harrison, 1952	1	0	Died with pulmonary embolism and c.n.s. disease 6 days later
Berk and Gerstle, 1952	1	1	Alive and well more than 8 years later
Baker, 1952	3	3	Alive and well 9 months to 4 years later
Schepel and Carsjens, 1953	1	1	Alive and well 2 months later
Susman, 1954	2	2	One alive and apparently well almost 5 years later, in spite of c.n.s. disease before resection; other alive and well 5 years later.
Poppe, 1954	2	2	Both alive and well 18 months later
Nichols and Martin, 1955	1	1	Alive and well 18 months later
Bonmati et al., 1956	4	3	Three alive and well 4 months to 2 years and 8 months later; one died of pneumonia and c.n.s. disease within a year later
Wettingfield et al., 1956	1	0	Died with small bowel obstruction, carcinoma of testis with metastasis, and pulmonary tuberculosis 5 weeks later
Pinney, 1956	1	1	Alive and well 1 month later
White and Arany, 1957	1	1	Alive and well 30 months later
Totals	26	21	

are apparently cured. The preceding table of results presents a range of apparent cures for periods of 1 month to more than 13 years.

2. Since 17 per cent of the reported resected cases of patients without prior central nervous system involvement subsequently developed it, the prognosis remains uncertain.

3. The most favorable prognosis is limited to cases in which there is no evidence of central nervous system involvement before excision of the localized pulmonary lesion; 90 per cent of the apparently cured survivors fall under this category.

4. A cure cannot be claimed justifiably unless the patient has remained well and without roentgenographic evidence of recurrence for at least 4 years following resection.

5. The early surgical excision of a localized pulmonary lesion before the onset of central nervous system dissemination can be regarded not only as a preventive, but also as a curative procedure.

6. The early adequate surgical excision of localized pulmonary cryptococcosis without central nervous system involvement appears to be the only effective treatment now known for this disease.

ADDENDUM

More recent communication reveals that all three resected cases reported by Ian McConchie of Australia, in 1951, remain alive and well. Among these, the man who had a left lower lobectomy, in 1940, represents the longest reported known survivor or cure, 17 years. The woman who was treated by excision of the pulmonary lesion and the adjacent chest wall in the presence of cryptococcal meningitis recovered and remains well 6½ years later. The third patient is alive and well 8 years after left lower lobectomy. The number of patients who have already remained well 4 years or more following pulmonary resection among the 21 reported survivors is, therefore, raised to at least 9.

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A STAINLESS STEEL BUBBLE OXYGENATOR

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THERE are three main types of extracorporeal blood oxygenators: membrane,[2, 5] film,[1, 3, 6, 8, 9] and bubble.[4, 7, 10] We wish to record a bubble type oxygenator which is made entirely of stainless steel. The advantages are in refinements. It can be entirely assembled before autoclaving, it is indestructible, and it does away with the debubbling heating helix. It can efficiently oxygenate volumes of blood from 100 c.c. to 4,000 c.c. per minute while adequately removing carbon dioxide to maintain the pH in a safe range.

DESCRIPTION

The oxygenator is constructed from two stainless steel tubes joined in a common debubbling chamber (Fig. 1). The length of these tubes is 100 cm., and the oxygenator tube is 1⅜ inches in inside diameter. The blood is loaded through a side vent, which is ½ inch in inside diameter, for gravity feed, or it can be made ¼ inch for pressure feed. The oxygenator manifold is a stainless steel disc containing 250 holes. These openings are .0024 inch in diameter. The filter manifold is a funnel-shaped disc of stainless steel which will act as a seat for the stainless wire mesh blood filter of the Baxter blood-giving sets.

The debubbling chamber is a 4,000 c.c. stock gauze container (Fig. 2). A fitted wire basket holding stainless steel sponges is inserted. No debubbling substances are necessary to make the removal of carbon dioxide adequate.

The second vertical tube is 1 inch in inside diameter and, functionally, goes only to the bottom of the debubbling reservoir, at which point four openings have been placed. This tube is a blood reservoir in which the blood is heated by a flood lamp to maintain a normal temperature. The blood hold-up in this tube is 500 c.c. Blood is pumped from the bottom of the manifold chamber after passing through the filter to return to the patient. These two tubes, although having different inside diameters, are interchangeable in their seats in the debubbling chamber.

The oxygen manifold chamber and filter manifold chamber are interchangeable (Fig. 3). Thus, blood can be oxygenated in the short tube with small inside diameter or in the larger, longer tube in large volumes.

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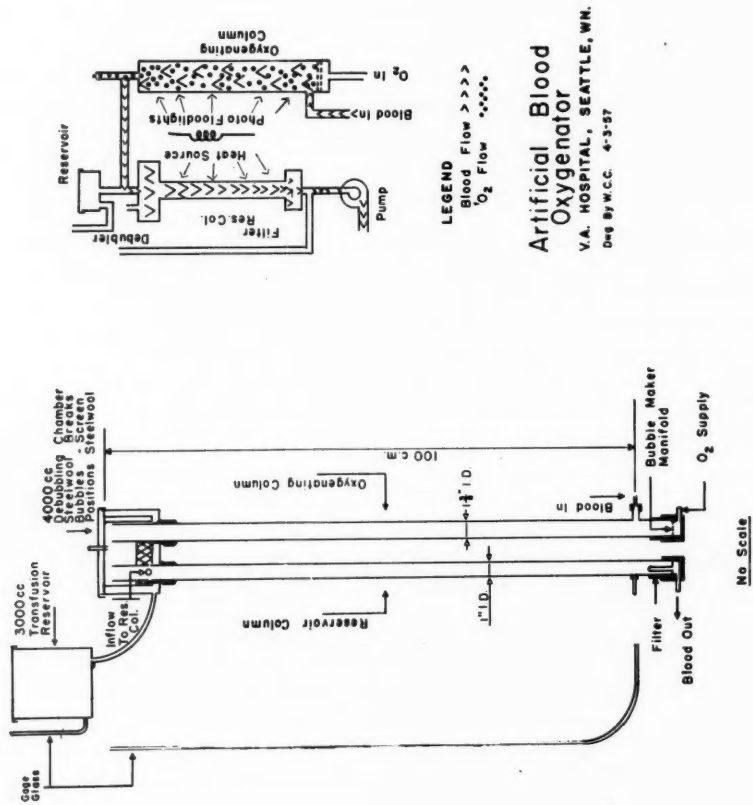

Fig. 1.—*A*, Oxygenator. *B*, Diagram of oxygenator.

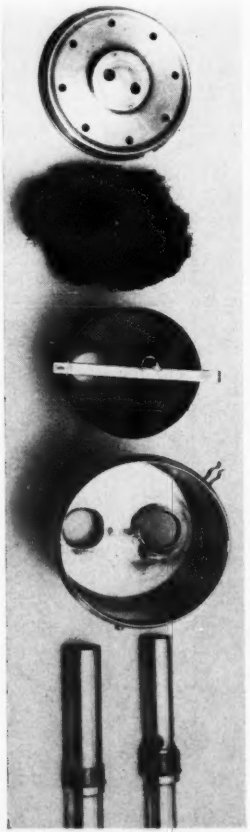

Fig. 2.

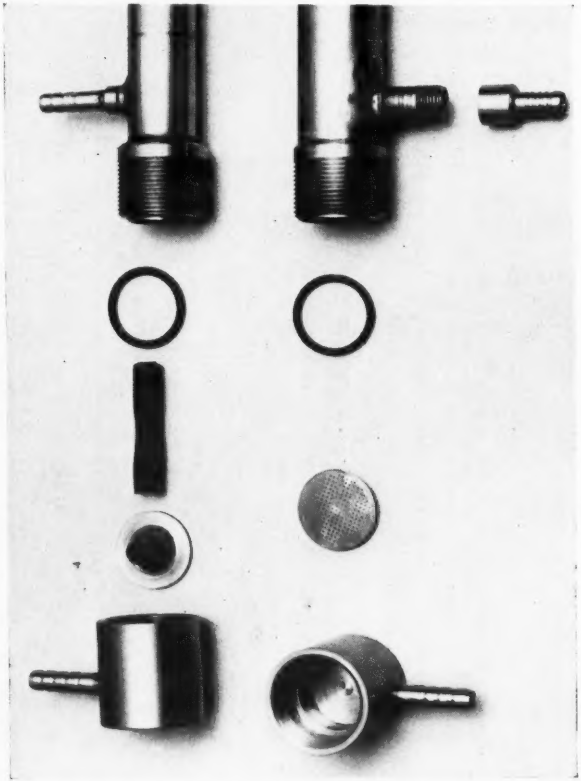

Fig. 3.

Fig. 2.—Debubbling chamber. *From bottom to top*—Oxygenating and reservoir tubes, debubbling chamber, stainless steel mesh holder, stainless steel mesh, and cover.

Fig. 3.—*Right side, from bottom to top* —Oxygen manifold chamber with side arm for oxygen inlet, oxygen manifold, "O" ring —washer, and oxygenating column and side arm with reduction nipple.

Left side, from top to bottom—Reservoir tube, with side tube for sight tube attachment, "O" ring— washer, stainless steel filter, filter manifold, and filter chamber with side arm for blood withdrawal.

The level of blood in the blood reservoir tube can be seen in the sight glass (Fig. 1).

A stainless steel blood reservoir of 3,000 c.c. is connected to the bottom of the debubbling chamber by plastic tubing for rapid transfusion. This reservoir can be raised or lowered to either transfuse or bleed the system.

A suction tube nipple is provided in the top of the debubbling chamber. Thus, blood of the suction flow is not lost but retransfused into the oxygenator.

PERFORMANCE

This oxygenator oxygenates at different minute outputs adequately (Table I).

TABLE I

EXP. NO.	RATE OF FLOW /MIN. C.C	HEMOGLOBIN		OXYGEN SATURATION IN VOLUME PER CENT		OXYGEN SATURATION PER CENT	
		VENOUS	ARTERIAL	VENOUS	ARTERIAL	VENOUS	ARTERIAL
1	500	17.30	17.10	7.58	22.34	32.21	94.61
2	1,000	17.60	17.25	13.84	21.92	57.84	92.69
3	1,500	17.61	17.35	12.42	23.51	52.67	99.18
4	2,000	17	16.75	18.13	22.79	78.71	99.24
5	3,000	16.45	16.5	17.98	21.63	80.67	95.57

The carbon dioxide loss as reflexed in the pH during perfusion can be shown to be adequate (Table II).

TABLE II

DOG NO.	TIME IN MINUTES OF PERFUSION	DOG BEFORE RUN		OXYGENATOR BEFORE RUN	OXYGENATOR	
		VENOUS	ARTERIAL		VENOUS	ARTERIAL
59	0	7.44	7.53	7.40		
	10				7.44	7.50
	25				7.45	7.54
	35				7.46	7.52
	45				7.44	7.52
	60				7.42	7.49
	73				7.45	7.52
58	0	7.46	7.56			
	5				7.45	7.55
	15				7.46	7.55
	19				7.44	7.54
57	0	7.60	7.71	7.39		
	5				7.43	7.64
	10				7.53	7.61
	15				7.52	7.61
	20				7.51	7.61
53	0	7.45	7.49			
	6				7.44	7.49
	14				7.40	7.49
	25				7.39	7.49

All values represent pH.

The effects on the formed and unformed elements of the blood are detailed in Table III.

TABLE III

20-30 MINUTE BY-PASS

	EXP. NO.	DOG, BEFORE BY-PASS	OXYGENATOR, BEFORE BY-PASS	DOG, END OF RUN	OXYGENATOR, END OF RUN
Platelets	1	337,000	50,000	150,000	150,000
	2	300,000	60,000	150,000	
	3	290,000	70,000	150,000	160,000
	4	184,000	94,000	88,000	88,000
Free hemoglobin mg./100 c.c.	1	23	65	56	46
	2	80	112	32	32
	3	4	4	170	170
Hematocrit	1	48	44	46	
	2	44		52	48
	3	44	41	52	48
	4	42	45	44	43
	5	41	45	47	45
White blood count	1	9,700	8,300	9,000	11,500
	2	8,200	8,600	9,000	9,500
Fibrinogen mg./100 c.c.	1	280	260	216	210
	2	340	290	230	330
	3	260	240	160	190
	4	250	217	240	240

CONCLUSION

A simple two vertical tubed oxygenator constructed completely of stainless steel is reported. It meets the requirements of simplicity with adequate performance.

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THE USE OF A MODIFIED GIBBON-TYPE PUMP-OXYGENATOR

I. Description of Apparatus

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THE successful employment of a screen-type oxygenator for use in an extracorporeal circulation has been demonstrated by Gibbon[1] and Kirklin.[2, 3] However, the high cost and engineering problems related to the construction of the model they employ have been a deterrent to its general use. Because of these factors, a modified version of the "Gibbon" stationary battery-type screen oxygenator has been constructed in such a manner as to eliminate costly and difficult fabrication methods without significantly altering its oxygenating function.

DESCRIPTION OF APPARATUS

Basically, the unit resembles that described by Gibbon[1] (Fig. 1). The top chamber (*A*) is constructed of Plexiglas in a pyramidal shape, its inside dimensions measuring 7.4 by 30.4 cm. with a height of 1.25 cm. The blood enters this chamber through inlet *B*. Tube *C* allows air to be removed from the chamber during filling, evacuation of any air which may enter the system during perfusion, and the addition to the system of any drugs desired. Soft rubber gaskets are placed on both sides of the plastic top chamber and the whole unit bolted through a rectangular steel cover, allowing securing of the chamber to the floor and tightening of the floor to the outside case.

The floor of the chamber is formed by 9 stainless steel bars which extend beyond the plastic top chamber, the 2 outer bars, *D*, measuring 2.5 by 37.5 cm., the 7 inner bars, *E*, 0.9 by 37.5 cm. The top and sides of these bars are machined flat, the bottom surface is shaped to form an inverted "V" along their length. The screens are attached along the length of the bar to one side of this "V" (Fig. 2).

The 8 stainless steel screens, *F*, measuring 30.5 by 55 cm. are fabricated from Tyler No. 538 screen.† The greatest dimension of the rectangular mesh in the screens is oriented in the horizontal plane. The screens are supported on the sides and bottom by 5 mm. stainless steel rods welded with silver solder.

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†Tyler Wire Company Cleveland, Ohio.

The top edge is held flush with the lower edge of the inverted "V" in the bottom of the bars by a 5 mm. stainless steel rod welded into the base of the "V," the edge of the screen being bent to fit between it and the bar. In assembling the unit, stainless steel shims measuring 0.15 mm. (0.006 inch) in thickness are placed at each end of bars, providing a 0.15 mm. by 30.5 cm. slit for blood to be admitted from the top chamber to the screens. The shims and bars are bolted together by 2 rods at each end. The use of shims between the bars

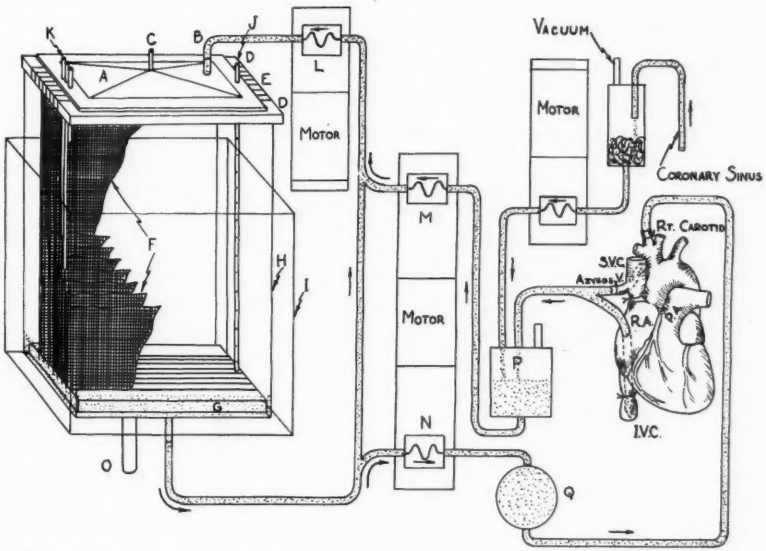

Fig. 1.—Schematic diagram of the pump-oxygenator. See text for description.

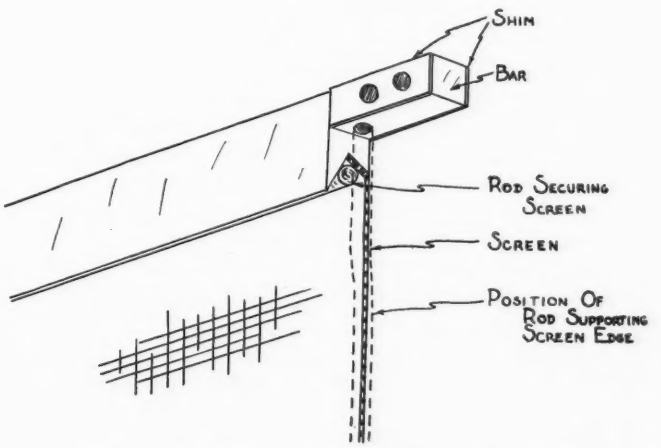

Fig. 2.—Diagram of bar and method utilized to attach screens.

permits the use of any number of screens desired, depending on the flow rate through the oxygenator. Those not in use are blocked by a single shim extending the length of the bar.

The base of the screens is placed in a Plexiglas block, *G*, the edges fitting into machined slots. The block is constructed so that it almost fills the internal dimensions of the base of the outside case, *H*, leaving approximately ⅛ inch clearance on all sides so that minute changes in blood volume in the reservoir produce significant changes in the height of the blood. One oxygen inlet tube, *J*, extends the length of the screens at one side of them and fits into the plastic block below while 3 oxygen outlet tubes, *K*, are placed at the opposite side; their length is such that oxygen flows out of the top, middle, and bottom of the unit. The tubes are press-fitted through the top bars.

The outside case, *H*, is constructed of ¼ inch Plexiglas, its dimensions such that it just allows insertion of the assembled screens, oxygen tubes, and base block, the bars resting on its top edge. A ¼ inch I.D. blood outlet tube and a ¾ inch O.D. saline outlet tube, *O*, are provided in the bottom of the case. A second case, *I*, is constructed outside case *H* to provide a thermostatically controlled water bath for constant control of the blood temperature and to prevent condensation of water vapor on the interior of the case.

Sigmamotor finger pumps* are utilized rather than the modified DeBakey pumps used in the originally described oxygenator. These pumps provide a pulsatile rather than a constant flow. Their speed is controlled manually with a vernier gear control.

These pumps are placed in relation to the oxygenator as shown in Fig. 1, pump *L* acting as a cycling pump for the oxygenator, and pumps *M* and *N* as venous uptake and arterial output pumps, respectively. A plastic venous reservoir (*P*) placed approximately 3 feet below the level of the right auricle receives blood by gravity flow from the venae cavae. Mayon plastic tubing† and stainless steel connectors are used throughout except for the short segments of soft latex tubing placed in the finger pumps.

The filter *Q* is constructed of two pieces of Plexiglas with fine mesh nylon cloth‡ held across the cavity between them. With the inflow placed at the top and the outflow at the bottom, the filter also acts as an air bubble trap.

This entire unit for extracorporeal circulation can be constructed and assembled for a cost of approximately $3,000.00, and eliminates many of the difficulties encountered in constructing the originally described models. Flow rates of 400 c.c. per minute for each screen can be obtained. This will give a total flow of 3.2 L. per minute if all 8 screens are used.

In operation, the entire unit is initially filled and washed with physiologic saline. The single Sigmamotor pump *L* which functions as the circulating pump for the oxygenator is used to fill the case completely with saline, its rate set to exceed the anticipated perfusion rate for the patient and to keep the top chamber filled, but not under pressure. The saline is then slowly

*Sigmamotor, Inc., Middleport, N. Y.
†Mayon Plastics, Minneapolis, Minn.
‡A. G. Bedin, Inc., Chicago, Ill.

emptied from the bottom outlet, the circulating pump being left on to continue the filming of saline on the screens until blood is added to the oxygenator.

Heparinized blood is added to the system through the venous reservoir, the remaining saline being emptied as the blood film forms on the screens and reaches the bottom of the oxygenator. The remainder of the system is then filled with blood, displacing the saline. Approximately 1,600 c.c. is required to prime the system, distributed as follows: 85 c.c. in the top chamber of the oxygenator, approximately 800 c.c. on the screens, 300 c.c. in the arterial reservoir in the bottom of the case, 300 c.c. in the venous reservoir, and 100 c.c. in the tubing and filter.

As soon as the blood is added and before placing the animal on the unit, a mixture of humidified 97 per cent oxygen and 3 per cent carbon dioxide is started at 10 L. per minute through the oxygen inlet tube.

METHOD OF PROCEDURE

Donor blood in sufficient quantity to prime the unit and replace expected blood loss is obtained for dog experiments by femoral cannulization after paralyzing the donor animal with 20 to 30 mg. of succinylcholine, and supporting respiration with intermittent endotracheal insufflation of room air. Heparin, 50 mg. per liter, aqueous penicillin, 1,000,000 U. per liter, and streptomycin 0.5 Gm. per liter are added at the time of bleeding.

Animals to be placed on extracorporeal circulation are prepared preoperatively by administration of morphine sulfate, 1.5 mg. per kilogram, atropine, 0.03 mg. per kilogram, and procaine penicillin, 600,000 U., given 45 minutes before the procedure is started. Pentothal sodium, 2.5 per cent, is administered intravenously for anesthesia and intermittent endotracheal insufflation of room air is utilized to maintain respiration throughout the procedure.

A routine right thoracotomy is performed, entering the pleural cavity in the fifth intercostal space. Meticulous care is taken to ligate all bleeding vessels. Tapes are placed about the superior and inferior venae cavae adjacent to their entry into the right atrium to be used for occlusion later. The right common carotid artery is isolated via an incision in the neck. A plastic catheter is passed into the carotid artery centrally. The superior and inferior venae cavae are also cannulated with plastic catheters inserted into the azygos vein and right auricle, respectively. Blood samples for pH, oxygen, and carbon dioxide contents are obtained at this time, at intervals during the procedure, and just after stopping the extracorporeal circuit.

The extracorporeal unit is then promptly connected to the animal, making certain no air bubbles are trapped in connecting the arterial cannula to the arterial outflow tube.

The arterial and venous pumps are now started, their flow rates previously set at 60 c.c. per kilogram per minute. The venous pump is speeded, maintaining the level in the venous reservoir until maximum venous return is obtained. Coincidentally the arterial pump is speeded, keeping the level of the

blood in the bottom of the oxygenator constant. This procedure is a relatively simple one since the large plastic block in the base of the case results in rather marked changes in the level with relatively small changes in volume. With proper adjustment of the pumps, the venae cavae are occluded by sliding rubber tubing tightly over the tapes and clamping. In most instances, endotracheal respiration is stopped during the perfusion. Blood may be returned to the venous reservoir and subsequently to the extracorporeal circuit by means of the coronary sinus system as illustrated in Fig. 1. Any blood loss is manifested by a fall in the level of blood in the venous reservoir.

The pH is followed throughout the perfusion and acidosis partially or completely corrected by the addition of 7.5 per cent sodium bicarbonate, usually in about 50 c.c. amounts. Protamine in amount equal to 1½ to 2 times the amount of heparin employed is then administered directly into the right auricle or very slowly intravenously during closure of the chest.

Both endotracheal and chest tubes are left in place until the animals show definite lightness of anesthesia, usually about one hour after the perfusion is terminated. In some of the animals with both weight loss and a fall in hematocrit after the procedure, citrated blood is given intravenously in adequate amounts.

During the initial runs of dogs on this pump oxygenator, during which time modifications were being made, the mortality was significant. With the final model as described above, 8 dogs have been operated upon. These animals had a right ventriculotomy and were maintained on the pump-oxygenator for 30 minutes. Of these dogs, 7 survived. The eighth dog was given an intravenous dose of protamine sulfate, penicillin, and streptomycin about one hour postoperatively. Shortly after this, it developed shock and died. A review of the physiologic changes in the experimental animals will be reported separately.

SUMMARY

A modified Gibbon-type pump-oxygenator has been described. This model functions well at high flows and can be constructed at a low production cost.

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